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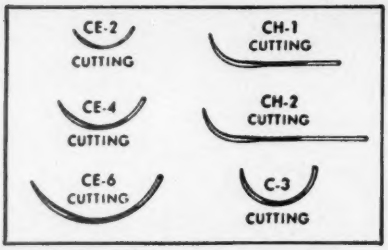

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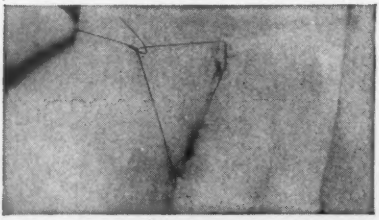

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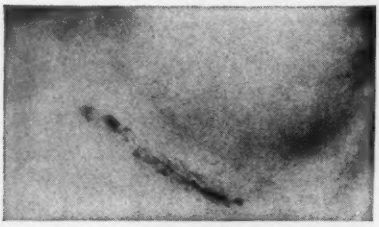

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SURGICAL MANAGEMENT OF MASSIVE GASTROINTESTINAL HEMORRHAGE

A REPORT OF THIRTY-NINE CASES

GEORGE A. OLANDER, M.D.*

Highland Park, Ill.

It is the policy of the Veterans Administration Hospital, Hines, Illinois, to admit patients with gastrointestinal bleeding to the Medical Service for their initial study and diagnosis. Surgical consultation is sought only when it is thought that medical management will not be successful. A summary of the cases treated by the Medical Service during the period involved in this study is presented. The summary gives the etiology, number of cases and mortality rate of patients with gastrointestinal hemorrhage (table I).

This series of cases parallels other similar series reported, and it emphasizes the fact that the majority of patients with duodenal ulcers respond favorably to medical management.

The 39 cases reported are those of patients operated upon by one surgeon and do not include patients seen in consultation and not operated upon. These patients were operated upon consecutively. The information obtained from observing these patients is included in the discussion. Although these patients were operated upon by one surgeon, their treatment depended upon the cooperation of all departments of the hospital and the diligent work of the residents.

This paper presents an analysis of the 39 patients operated upon including the mortality rate and complication statistics, table II and table III. Following this analysis, a discussion is presented of the important factors in the diagnosis, preoperative and postoperative therapy, indications for surgery, anesthesia and

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surgical technic. This paper is not written to discuss the pros and cons of surgery on patients with gastrointestinal bleeding nor to present an extensive literature review. The comments made point out the features of interest in the individual cases. The factors that have been found to be of importance in the care of these patients that are not ordinarily discussed are presented.

Only the major and significant complications are listed. Those that occurred in the patients who died are not included but the significant autopsy findings are presented. There were proportionately more patients with gastric ulcer operated upon than patients with duodenal ulcer.

Diagnosis. The diagnosis of the pathology and type of massive gastrointestinal hemorrhage is very important if proper therapy is to be instituted. The foregoing statistical analysis illustrates this point. Patients with bleeding gastric ulcer

TABLE I

Bleeding Site	Number of Cases	Deaths	Per cent
Duodenal ulcer	244	4	1.64
Cause undetermined	71	5	7.04
Esophageal varices	26	16	61.54
Gastric ulcer	21	3	14.28
Gastritis	21	0	0
Prolapsed gastric mucosa	7	0	0
Marginal ulcer	6	0	0
Hiatus hernia	4	0	0
Esophageal ulcer	2	0	0
Carcinoma esophagus	1	0	0
Carcinoma stomach	1	0	0
Carcinoma pancreas	1	1	100
Thrombocytopenia	1	0	0
Bleeding gastric ulcer	1	0	0

have a higher mortality rate on conservative therapy than would be expected from early surgical treatment. Patients who had a long history of duodenal ulcer, and who previously had a roentgenographic diagnosis of duodenal or gastric ulcer, or who previously bled and the site of bleeding was established, present fewer problems of diagnosis than patients who have never had bleeding or who have no history of ulcer. It is the latter group of patients who present the greatest diagnostic problems and about which the following comments are made.

Patients with esophageal varices present a particular problem of management because their bleeding episodes sometimes can be managed by the use of the Sengstaken tube. Two patients in the group had esophageal varices as the source of their bleeding and were misdiagnosed prior to operation. Both of these patients were young men who appeared to be healthy. Neither presented the typical history of a patient with cirrhosis of the liver. One of them—case 33—had a history of transitory jaundice while in the Army in North Africa. This was the only indication in the patient's history or physical examination that indicated possible liver pathology. The other patient—case 34—had a history of previous

hospital admission because of a marked dietary deficiency. These two points in the history—although appearing to be minor—might justify the consideration of esophageal varices as the cause of bleeding and the use of a Sengstaken tube as a diagnostic aid. The use of this tube will be discussed later. This patient—case 34—also had a history of sudden acute abdominal pain associated with a rigid abdomen. Roentgenographic examination was negative for free air in the abdomen, but it was thought that the patient had a bleeding and perforated ulcer. The cause of the abdominal rigidity was a rupture of the esophagus. We have had 2 other cases of rupture of the esophagus, excluding the one with partial rupture, and both presented the signs and symptoms of a perforated peptic ulcer. The rupture occurred in all of these patients following a vigorous vomiting episode.

TABLE II

Summary of cases

Duodenal ulcer	19	Pulmonary edema	2
Gastric ulcer	11	Serum jaundice	2
Bleeding site unknown	3	Incisional hernia	2
Varices	2	Pulmonary infarct	1
Complications		Rupture of esophagus	1
Wound separation	5	Thrombocytopenia	1
Thrombophlebitis	4	Vessel in fundus	1
		Postgastrectomy bleeding	1

Deaths

Case	Cause of Death
17	Lower nephron nephrosis, peritonitis (chemical), toxic hepatitis, focal pancreatic necrosis.
18	Hepatitis, bronchopneumonia.
34	Rupture of esophagus, empyema, brochopneumonia, esophageal varices.
37	Massive pulmonary infarct.

Roentgenographic examination of the mediastinum for air, or after a swallow of Umbrathor may aid in the diagnosis.

Gastrointestinal hemorrhage due to blood dyscrasia is also an important differential diagnosis if proper therapy is to be instituted. One patient—case 38—was treated on the Medical Service and referred to surgery for massive gastrointestinal hemorrhage. The gastrointestinal tract was the only site of bleeding. He did not have visible petechial hemorrhages. However, in this patient's history, abnormal bleeding was specifically noted following a tooth extraction a few years previously. For this reason, complete blood studies were made and it was found that he had thrombocytopenia. He responded to splenectomy.

A history of recurrence of hematemesis or melena, and color and quantity of each may be helpful in determining the site of bleeding. Patients with esophageal varices usually have bright red emesis of blood accompanied by melena as do patients with gastric and duodenal ulcer. However, this history can be very

TABLE III

Summary of cases

Case #	Bleeding Site	Age	Ulcer Hist.	Duration Bleeding	Blood Preop.	Blood dur. Surg.	Preoperative Complications	Indications for Operation	Operation	Postoperative Complications
1	Duodenal ulcer anterior wall	56	Yes	3 days	8,000	1,000	None	Shock, persistent hemorrhage	Excision of ulcer, gastric resection	None
2	Duodenal ulcer second portion	61	Yes	6 Days	4,000	1,000	Glaucoma, hypertension, diabetes, cirrhosis	Shock, confusion, persistent bleeding	Ligation of vessel, gastric resection	Incisional hernia
3	Duodenal ulcer greater curvature	23	Yes	5 Days	5,000	3,000	None	Previous bleeding, massive hemorrhage	Gastric resection	Pulmonary edema, homologous serum jaundice
4	Posterior duodenal ulcer	28	Yes	2 Days	8,000	1,500	None	Previous bleeding, massive hemorrhage	Gastric resection	None
5	Duodenal ulcer anterior and lateral aspect	31	Yes	1 Day	3,500	1,500	None	Massive hemorrhage on medical management	Gastric resection	None
6	Duodenal ulcer lateral aspect	30	Yes	1 Day	3,500	500	Plication of small bowel	Two previous hemorrhages, massive hemorrhage	Ligation of right gastric artery supplying ulcer	None
7	Duodenal ulcer anterior surface	70	Yes	2 Days	6,000	1,000	Cirrhosis	Shock, massive hemorrhage	Gastric resection	None
8	Posterior duodenal ulcer	55	Yes	2 Weeks	7,000	1,000	Gastric obstruction, arteriosclerosis	Shock, persistent bleeding, gastric obstruction	Gastric resection	Wound separation
9	Ulcer, posterior duodenum, second portion	32	Yes	3 Days	5,500	2,000	None	Shock, massive hemorrhage	Ligation of vessel, gastric resection	Thrombophlebitis
10	Duodenal ulcer adherent to gallbladder and invading right gastric vessel	37	Yes	3 Days	8,500	2,000	None	Shock, massive hemorrhage	Gastric resection	None
11	Posterior duodenal ulcer	29	No	2 Days	9,000	1,500	None	Massive hemorrhage, shock	Gastric resection	None
12	Posterior duodenal ulcer	53	Yes	1 Month 2 Days Acutely	4,000	2,000	Previous bleeding	Previous hemorrhage, shock	Gastric resection	None
13	Anterior lateral duodenal ulcer	30	Yes	3 Days	5,000	1,000	None	Massive hemorrhage	Gastric resection	None
14	Anterior duodenal and gastric ulcer	57	Yes	3 Days	12,000	1,000	Hemiplegia, prostatic hypertrophy	Massive hemorrhage	Gastric resection	None
15	Posterior duodenal ulcer	37	Yes	4 Days	6,000	1,500	Pyloric obstruction	Massive hemorrhage	Gastric resection	None
16	Anterior duodenal ulcer	28	Yes 6 yrs.	5 Days 2 episodes	5,000	1,500	None	Massive hemorrhage. Bled on medical management	Gastric resection	None
17	Posterior duodenal ulcer	58	Yes	7 Days	6,000	1,000	Hypertension Parkinson's disease	Shock, massive hemorrhage	Gastric resection	Died
18	Posterior duodenal ulcer	43	Yes	2 Days	6,500	1,500	None	Previous hemorrhage, shock, massive hemorrhage	Gastric resection	Died
19	Duodenal ulcer involving pancreaticoduodenal artery	65	Yes	10 Days	5,000	1,500	Shock, taboparesis	Massive hemorrhage roentgenographic evidence	Gastric resection	None

18	Posterior duodenal ulcer	43	Yes	2 Days	6,500	1,500	None	Previous hemorrhage, shock, massive hemorrhage	Gastric resection	Died
19	Duodenal ulcer involving pancreaticoduodenal artery	65	Yes	10 Days	5,000	1,500	Shock, taboparesis	Massive hemorrhage roentgenographic evidence	Gastric resection	None
20	Lesser curvature gastric ulcer	64	Yes	6 Days	3,000	1,250	None	Massive hemorrhage roentgenographic evidence	Gsatric resection	None
21	Antral gastric ulcer	37	Yes Acute	5 Days	3,500	500	None	Shock	Gastric resection	None
22	Lesser curvature ulcer, 1.5 cm.	58	Yes	9 Days	13,500	1,500	Obesity, bronchopneumonia	Massive hemorrhage	Hoffmeister gastric resection	None
23	Lesser curvature ulcer	37	Yes	1 Day	3,500	1,000	None	Hemorrhage on medical management	Gastric resection	None
24	Lesser curvature ulcer	32	Yes	4 Days	3,000	1,000	None	Shock	Gastric resection	None
25	Lesser curvature ulcer, 6 cm.	57	Yes	1 Day	5,000	1,500	None	Shock, bled on medical management	Hoffmeister gastric resection	Thrombophlebitis
26	Lesser curvature ulcer, 4 by 2 cm.	57	Yes	4 Days	11,000	2,000	None	Massive hemorrhage	Hoffmeister gastric resection	Pulmonary infarct, thrombophlebitis
27	Lesser curvature ulcer	53	Yes	7 Days	10,000	2,000	Carcinoma antrum	Massive hemorrhage	Anterior Hoffmeister resection	Wound separation
28	Lesser curvature ulcer	37	Yes	2 Days	5,000	1,000	None	Massive hemorrhage	Posterior Hoffmeister resection	Wound separation
29	High lesser curvature ulcer	49	Yes	2 Days	5,000	1,000	None	Shock, massive hemorrhage	Posterior Hoffmeister resection	None
30	Lesser curvature ulcer, 1.5 cm.	64	No	1 Day	8,500	2,000	Pulmonary emphysema	Massive hemorrhage	Gastric resection	Homologous serum jaundice, wound separation
31	Partial rupture esophagus	26	No	12 hrs.	5,000	500	None	Massive hemorrhage	Gastrotomy, suture of esophagus	None
32	Ligation of vessel in fundus of stomach	33	No	7 Days	8,500	2,000	Brucellosis	Previous hemorrhage, massive hemorrhage	Gastrotomy, ligation of vessel	None
33	Varicosities of esophagus	31	No	7 Days	6,500	2,500	Cirrhosis	Massive hemorrhage	Laparotomy	Splenorenal shunt accomplished at later date
34	Varicosities, rupture of esophagus	38	No	1 Day	2,500	1,000	Cirrhosis	Hemorrhage and acute abdomen, misdiagnosed	Gastrotomy, Snegstaken tube inserted	Died pulmonary sepsis
35	Not found, x-ray evidence of duodenal ulcer	25	No	7 Days	5,000	1,500	Obesity	Continual bleeding	Gastrotomy pulmonary edema developed	None Pulmonary edema
36	None found, x-ray suggestive of ulcer	47	Yes	1 Day	5,500	500	Obesity	Massive hemorrhage	Gastrotomy	Wound separation
37	None found at surgery or autopsy, x-ray suggestive of duodenal ulcer	26	Yes	2 Days	3,000	1,500	None	Massive hemorrhage	Gastric resection	Pulmonary infarct. Died
38	Thrombocytopenia gastrointestinal mucosa	52	Vague	6 Days	10,000	500	None	Continued hemorrhage	Splenectomy	Incisional hernia
39	Bleeding from gastrectomy suture line	57	Yes	1 Day	3,000	1,000	Gastrectomy	Continued bleeding evisceration	Gastrotomy ligation of vessel	None

misleading in some cases. Two patients—cases 15 and 16—had duodenal ulcer associated with pyloric obstructions. They never vomited blood. One patient—case 30—had a gastric ulcer on the lesser curvature of the stomach. He did not vomit blood at any time during his bleeding episode.

The physical examination of the patient should be complete. The factors that are particularly important in reference to massive gastrointestinal hemorrhage are petechial hemorrhage, a palpable spleen and liver, a stomach distended with blood and the activity of the small bowel.

Any patient with petechial hemorrhage should have complete blood studies for possible blood dyscrasia as the cause of his bleeding. The presence of a palpable spleen or liver should make the surgeon suspicious of esophageal varices as the cause of bleeding even though there are no other signs of portal hypertension. Because of abdominal distention—as a result of blood in the gastrointestinal tract—the spleen may not be palpable. This was found in the 2 patients operated upon for esophageal varices. The diagnosis of a stomach distended with blood is important because such distention can interfere with respiration; it precludes the continuation of feeding therapy for bleeding ulcer and roentgenographic diagnosis is not accurate. A marked increase in small bowel activity may be one of the first signs of bleeding. The important features of the physical examination relative to fluid and blood replacement are discussed in preoperative preparation.

Roentgenographic examination of the gastrointestinal tract in the period between bleeding episodes may be very helpful in determining the site of bleeding. Such an examination was of aid in locating the bleeding site prior to operation in 9 of the 39 cases. These patients did not have a previous gastrointestinal roentgenographic series.

The procedure used at this hospital is as follows: the patient is placed on his back on the fluoroscopic table and given a few swallows of the barium mixture while roentgenograms are taken of the esophagus in right and left anterior oblique position. With a small quantity of barium, spot roentgenograms of the stomach are taken with the patient lying on his back, after rotating him with his left side up and then with his right side up. Pressure by hands or pressure cone is avoided. The remainder of the opaque meal is then given; the patient is turned on his abdomen; his back is marked for position; and larger roentgenograms are taken in the posterio-anterior, right oblique and lateral positions. The esophagus roentgenograms are then taken as desired. A five hour roentgenogram is not necessary in these cases. This procedure is carried out under the supervision of the doctor with an adequate intravenous transfusion in place; available blood and preferably with the stomach empty. Differentiation between a gastric and a dudodenal ulcer is an important factor in determining the course of treatment given a patient with gastrointestinal hemorrhage. This is evidenced in the mortality statistics of patients given medical treatment for duodenal and gastric ulcer.

The use of the Sengstaken tube as a diagnostic and therapeutic agent is very important in selected cases. In our experience, it would seem reasonable to recommend that all patients with a suggestive history or physical findings of liver or

spleen pathology should have a trial use of the Sengstaken tube prior to consideration of surgical correction of his bleeding. Although the Sengstaken tube is uncomfortable to the patient, it can be tolerated when the importance of its use is properly explained and vasoconstrictors and anesthetic agents are applied to the nose and pharynx. It is important to correctly use the Sengstaken tube. The gastric and esophageal balloon should be tested before the tube is inserted. The maximum capacity of the gastric balloon should be determined and this quantity of air should be injected into the gastric balloon after it is placed in the stomach. Gentle traction is then applied upon the tube and the esophageal portion of the ballooon is distended until it is maintained at about 30 mm. of mercury (fig. 1). Because of the construction of the Sengstaken tube, it has been

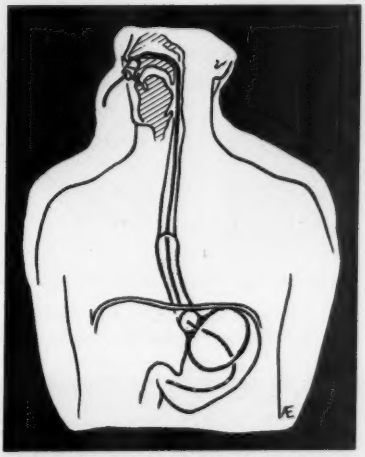

FIG. 1. Diagram of the Sengstaken tube in place. This illustrates how the gastric balloon can tamponade bleeding from a high gastric uler. It also illustrates that there is little tamponading effect at the esophageal cardiac junction.

misleading in the diagnosis of 2 cases. One patient had a high lesser-curvature gastric ulcer. After the Sengstaken tube was inserted, the patient stopped bleeding because of the tamponading of the gastric balloon against the gastric ulcer. Another patient—case 31—had a partial rupture of the esophagus at the esophageal cardiac junction. He continued to bleed after the Sengstaken tube was inflated because the esophageal cardiac junction did not have the tamponading effect of either the esophageal or gastric balloon due to the location of the lesion between the two balloons. When a patient with esophageal varices is incorrectly operated upon. the Sengstaken tube can be inserted at the time of operation and its direct effect upon control of the hemorrhage can be visualized.

Esophagoscopy as a diagnostic aid should not be forgotten. It was important in the differential diagnosis of 1 patient who had roentgenographic evidence of possible esophageal varices; an esophageal diverticulum; and a possible gastric ulcer. These lesions are shown in figures 2 and 3.

Preoperative Preparation. In the immediate preoperative period, the majority

FIG. 2. Esophagram demonstrating a low esophageal diverticulum and an irregularity of the lower esophagus that was interpreted as possible varices of the esophagus.

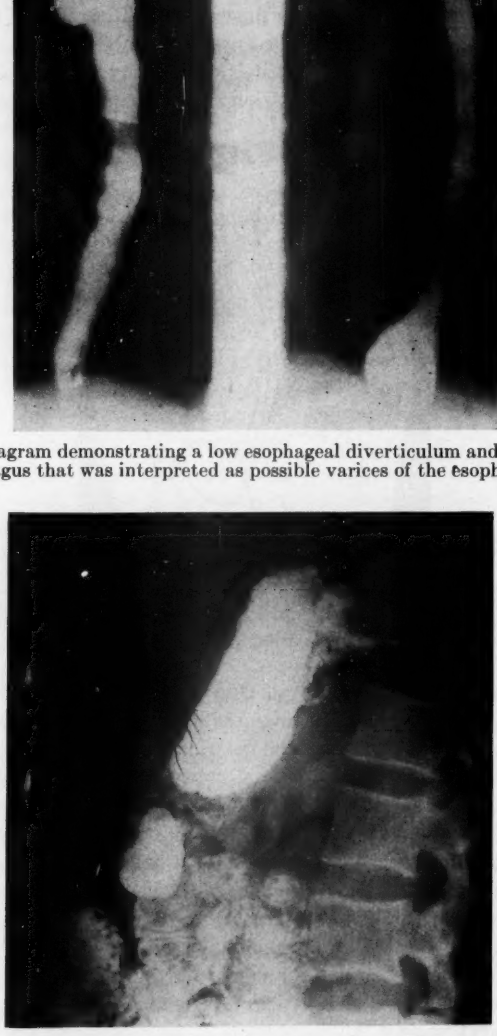

FIG. 3. Lateral view of the stomach of the same patient. The posterior projection of barium demonstrates the site of the gastric ulcer.

of patients were transferred to the recovery ward. During this time all of the advantages of the recovery ward were utilized to prepare these patients for operation. As was previously stated, this group of patients represents those considered to have had massive or sustained upper gastrointestinal bleeding. It is for this reason that adequate administration of blood is essential for the care of these patients. This procedure begins with proper typing and cross-matching of blood and ascertaining whether or not there is an adequate quantity of blood available. The availability of blood might seem to be no problem at Hines Hospital with an adequate blood bank, but it has been a factor in patients with rare blood types. Large needles—15 gauge—or large catheters should be inserted into the peripheral veins so that blood can be given continuously and rapidly if necessary. Pressure apparatus for the administration of blood should be available. If administration of blood under pressure is contemplated, the doctor should make certain that the intravenous equipment is adequate for this procedure. The best guide to the quantity and rate of blood to be administered is the blood pressure, the pulse, venous filling, pulmonary edema, sweating and mental confusion. These observations should be supplemented by laboratory examinations such as the blood count, hematocrit, and urine examination. However, the clinical response of the patient to bleeding and his response to transfusion are the most important signs. More extensive laboratory procedures may be indicated during the periods when the patient is not bleeding. While the patient is receiving blood a careful check as to the amount and quality of the fluids administered with the blood should be maintained. Physiologic sodium chloride solution should be kept at a minimum and usually the quantity necessary to maintain the blood transfusion is adequate. Five or 10 per cent glucose solution should be given to maintain an adequate urinary output and state of hydration. Vitamins C, B and K should be added to this fluid in therapeutic quantities. Because patients with hemorrhage and associated vomiting and diarrhea may develop a potassium deficiency, this factor should be ascertained by the accepted procedures and potassium should be given to the patient in therapeutic quantities.

Gastric Suction. The decision as to whether or not a patient should be fed or maintained on gastric suction is sometimes an important one. This decision will depend upon the site of bleeding, the rate of bleeding and the degree of gastric distention. Gastric dilatation should be minimized at all times. Patients should have a large Levine tube inserted prior to operation and the stomach evacuated as much as possible before the anesthetic and operation are started. At times this is impossible, if the rate of bleeding is intense. A Levine tube can be inserted during the operation and after the bleeding has been controlled. At present a double-lumen tube (fig. 4) is available which has adequate suction holes for removing liquid blood. This tube is preferred because it can be inserted past the gastroenterostomy, thus assuring adequate postoperative feeding. Obstruction of the gastroenterostomy stoma precipitated complications that resulted in the death of 1 patient—case 18.

Sedation. The majority of patients with massive gastrointestinal bleeding are very apprehensive. The presence of a tube in their pharynx may be irritating,

and if they have been allowed to go into shock, they may be restless and uncooperative as a result of cerebral anoxia. It is for this reason that the judicial use of sedation is important. Restlessness from cerebral hypoxia should be primarily treated by the administration of blood and nasal oxygen until the condition of the patient improves. The use of topical anesthesia is indicated in those patients who complain of throat discomfort from the tube.

Patients who are restless as a result of their apprehension are best treated by the administration of a narcotic such as morphine or demerol with reassurance and explanation as to the cause and course of their illness.

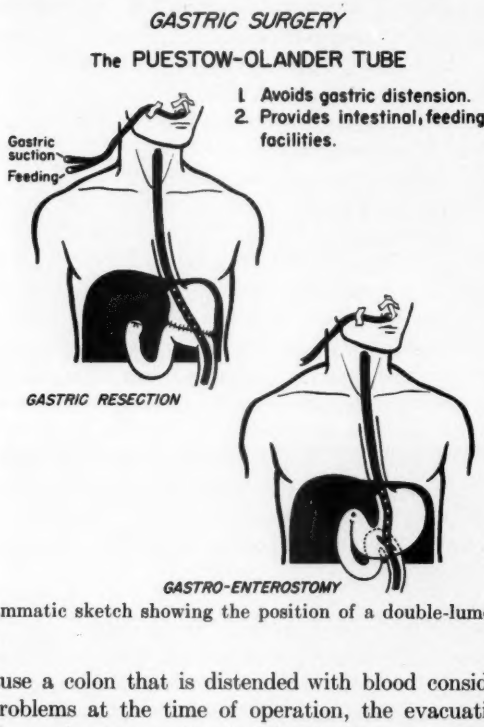

FIG. 4. Diagrammatic sketch showing the position of a double-lumen tube as used in gastric surgery.

Enema. Because a colon that is distended with blood considerably increases the technical problems at the time of operation, the evacuation of the colon with an enema just prior to operation is helpful. An enema is not indicated very often because the majority of patients with massive bleeding readily evacuate their colons.

Indications for Operation. The selection of the proper time to advise operation in the treatment of patients with hemorrhage still remains a major problem. The statistics previously presented indicate that the majority of patients respond favorably to conservative management. However, it is just as serious an error to procrastinate in the medical management of the patient with a resultant increase in mortality rate as it is to operate upon a patient when it is not indicated. Several authors have attempted to formulate rules to decide when to operate

upon patients with massive hemorrhage. In effect, they have defined massive hemorrhage and have used this as an indication for operation. The patients in the series listed as having massive hemorrhage fit into this category. That is, the red blood count fell precipitously to 2,500,000 per cu.mm. or less. They manifested signs and symptoms of shock and required 500 cc. or more of blood every eight hours to maintain their blood pressures and pulse rates at near normal levels.

There are several other factors that are important in deciding when to operate upon patients with massive hemorrhage. One of these is the patient's response to hemorrhage. For example, one patient may blood 500 or 1000 cc. of blood and respond very poorly to this hemorrhage by not only having the usual signs and symptoms of shock but may also become markedly confused and disoriented. Another patient with an equal mount of bleeding may not manifest as marked signs and symptoms of shock and may not become mentally confused. The first patient is more of a surgical emergency than the second because his chance of surviving repeated hemorrhages is much less than the second patient. Another group are patients who have other indications for operation such as duodenal ulcers, in addition to hemorrhage; those who have had multiple previous hemorrhages; signs and symptoms of obstructions; or who have had intractable ulcers for a long period of time. This group of patients should be considered for operation even though their bleeding is minimal, providing their general physical status would warrant a gastric resection or other definitive therapy for their ulcers. One of the patients in the series reported—case 17—falls into this group. This patient had eight previous hemorrhages. He entered the hospital with minimal bleeding; his surgery was delayed; and he then had to be operated upon as an emergency after he had multiple transfusions. His autopsy findings have been presented; and it is reasonable to assume that his death may have been prevented by earlier operation. Two other patients with multiple episodes of bleeding have died on medical management. Two patients that are in this category recovered uneventfully following operation.

One other group of patients who should be considered for early operation are those who bleed on medical management. They are patients who enter the hospital with persistent abdominal pain; are placed on strict medical management and during this therapy begin to bleed. In the series reported there were 2 patients in this category and, although their surgery was deferred, both of them survived.

Anesthesia. There is no question that successful anesthesia for these patients requires that the anesthesiologist not only thoroughly understand the pharmacology of all the anesthetic agents, but also that he is an expert technician. Delay of administration of drugs can be rapidly fatal. It is obvious that the patient must be as well prepared for operation as possible. Occasionally, however, patients have been operated upon who were in poor condition because of the rate of bleeding. We have required that six pints of blood be available before operation is started. We have not required that this quantity of blood be given during operation, but it is insurance against delay at a critical moment. Other safety

factors are the availability of several suction tubes for aspiration of the pharynx and trachea, bronchoscopy tray and sufficient assistance.

Premedication is kept at a minimum. Spinal anesthesia was never used. The majority of patients were intubated with a curare-pentothal induction. Cyclopropane was the most commonly used anesthetic agent.

Surgery. The majority of these patients were explored through a left paramedian incision. This incision gives adequate exposure. However, in a patient who is obese and has a small cascading stomach, an incision extending from the

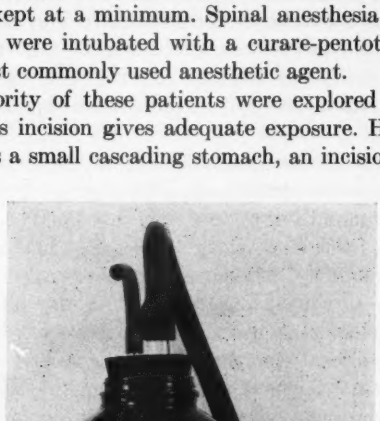

FIG. 5. Photograph of the suction bottle which has been used successfully in evacuating the stomach of large blood clots. The increased size of the suction tubing is illustrated.

left intercostal notch to point to the right of the umbilicus affords better exposure of the duodenum. This incision is helpful when closure of the duodenum is difficult. The majority of incisions were closed in the usual manner. However, patients with whom the anesthesiologist had difficulty controlling respirations, or those with poor tissue, a very rapid and adequate closure was obtained with interrupted figure of eight stainless steel wire sutures.

The principle aim in the surgical procedure is to control the source of bleeding. The secondary aim is a procedure that gives the patient the best possible chance of not having the bleeding recur. It is for this reason that the surgeon should not have a definitely planned procedure, but rather vary the surgical procedure to fit the individual case. Factors that are important in this decision are the general condition of the patient; his response to blood transfusion after

the bleeding has been stopped; the anatomy of the vascular tree; and the location of the pathology. It is essential that the stomach be evacuated of blood—if it is markedly distended—to facilitate the surgical procedure. This is best accomplished through a gastrotomy incision. The clots may be manually removed, but the suction bottle illustrated in figure 5 has been found to be more adequate. The large suction tube does not become clogged and it rapidly removes the blood. If the source of bleeding is not apparent or has not preoperatively been diagnosed, a planned procedure for locating the bleeding point is recommended. In such cases this was accomplished by completely evacuating the stomach of blood and blood clots and placing a dry laparotomy pad in the fundus of the stomach, and a smaller laparotomy pad or a free sponge in the duodenum. These were left in place for a period of time and separately removed. In this manner the area of bleeding, whether it was in the fundus of the stomach or the duodenum, could be readily ascertained. While these laparotomy pads remain in place, careful inspection of the small bowel may be made to rule out rare and unusual sources of bleeding from the small bowel.

All of the patients with duodenal ulcer—except 2—had a gastric resection—posterior Polya type—with excision of the ulcer as the surgical procedure for controlling their bleeding. One patient—case 6—who previously had two operations, one of which was a plication of the small bowel, had a duodenal ulcer on the lateral aspect of the duodenum involving the right gastric artery. The right gastric artery and its branches were ligated external to the duodenum which controlled the bleeding. The other patient—case 9—had a small duodenal ulcer on the posterior aspect of the duodenum in the second portion. The bleeding vessel was ligated through a duodenotomy incision and the duodenal stump was closed above it. In this group all of the duodenal stumps were closed by the open technic. In those ulcers that were low and inflammation involved the porta hepatis, the gallbladder was compressed and the free flow of bile into the duodenum was visualized to determine whether or not the common duct was injured. There was no common duct injury in this group. Some of the patients with a lesser curvature ulcer required a Hoffmeister type of resection.

Case 31 the patient had a partial rupture of the esophagus. This was sutured through a gastrotomy incision which controlled the hemorrhage. A Sengstaken tube with the gastric balloon inflated and wrapped with gelfoam was then tamponaded against the cardia. In another patient—case 32—all that was necessary to control the bleeding was ligation of a bleeding vessel in the fundus of the stomach. There was no evidence of ulcer or varices. The exact nature of this bleeding vessel was not determined.

Postoperative Care. In general the postoperative care of these patients was the same as for the routine gastric resection. The variations that have been found to be beneficial are adding fluids—one liter at a time—to avoid overloading. Blood is replaced in the same manner as during the preoperative period. A significant number of patients developed an anemia on the fifth to seventh postoperative day even though the blood count and hematocrit were normal immediately after operation. One other alteration that was commonly noted post-

operatively was a persistent blood pressure elevation for 24 to 72 hours. This blood pressure elevation was at times 30 to 40 mm. of mercury above the normal systolic pressure. No specific therapy was used to control this elevation. The blood pressure of all of these patients returned to normal.

SUMMARY

Thirty-nine consecutive cases representing patients having massive gastrointestinal hemorrhage operated upon by one surgeon are presented. The salient features pertinent to these cases and the errors that were made in the management are discussed.

ACUTE DIVERTICULITIS OF THE CECUM

A REPORT OF SIX CASES

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Diverticulitis of the cecum has been recognized as a disease entity for some time and has been reported with increasing frequency in the past decade. Potier[4] reported the first case in 1912. Schnug[6] in 1943, found 32 cases in the literature and added 6 cases from the Cincinnati General Hospital. Anderson[1] reviewing the literature in 1947, found 9 cases of acute diverticulitis of the cecum in *some 700 cases* of surgical diverticulitis of the colon at the Mayo Clinic, and brought the total number of reported cases to 99. Since that time, intermittent reports have brought the total of reported cases close to 160.

Diverticula of the colon are classified as *true or congenital* and *false or acquired*. The *true or congenital type* is found to occur singly although instances of more than one diverticulum have been reported. Usually this diverticulum is found within several centimeters of the ileocecal valve and histologically it shows all the layers of the normal intestinal coat. The *false or acquired type* is commonly multiple and is found most often in the sigmoid colon, but may be found in any portion of the large bowel between the ileocecal valve and the rectum. Its etiology is thought to be explained by the exertion of increased intracolonic pressure on potential hernia areas. In those areas, blood vessels pierce the muscular coat of the intestinal wall, allowing the mucosa to protrude through; therefore, histologically, this type is composed of only mucosa and serosa.

In more than half of the cases of cecal diverticulitis, *or the congenital* type, a fecalith is reported to be present, suggesting that the pathologic process is pressure necrosis followed by inflammatory reaction. In the large series of cases collected by Lauridsen and Ross[2] the average age of patients with cecal diverticulitis was 40.4 years for males and 40.8 years for females, although the ages ranged from 10 to 72 years. This type occurs with slight increased frequency in males. The *acquired type* characteristically occurs in males after 45 years of age. Of 144 cases of sigmoid diverticulitis treated by operation at the Mayo Clinic[3] the average age of the patients was 53.6 years, with a male preponderance of 2 to 1.

The symptoms and physical findings of congenital diverticulitis are usually indistinguishable from acute appendicitis. On comparing symptoms in a large series of cases of appendicitis with those of diverticulitis of the cecum, the significant difference was the percentage of patients experiencing nausea and vomiting. In appendicitis, nausea occurred in 78 per cent and vomiting in 73 per cent of the patients[5]. In cecal diverticulitis, nausea occurred in about 27 per cent and vomiting in about 16 per cent of the patients. Since the symptoms are so much

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alike, it is not surprising that the diagnosis of acute appendicitis is almost always made in those patients who have *not* had an appendectomy. In those who *have* had an appendectomy, the diagnosis is usually carcinoma, diverticulitis, abscess, or pelvic inflammatory disease.

The diagnosis made in cases of cecal diverticulitis at the time of operation often is incorrect. While it is true that approximately 70 per cent of such cases are

TABLE I

Operation	No. of Cases	Reporting Results	Died	
			No.	Per Cent
1. Excision	67	62	1	1.6
2. Colon resections	57	55	4	7.3
a. Right colectomy	34	32	3	9.4
b. Cecectomy	16	16	1	6.3
c. Ileocecal resection	7	7	0	0.0
3. Inversion	10	10	0	0.0
4. Drainage	8	8	1	12.5
5. Exteriorization	5	5	0	0.0
6. Other	6	6	1	16.6
Total	153	146	7	4.8

TABLE II

Fate in 39 cases with incorrect diagnosis of cancer at operation

Operation	Cases	Mortality	
		No.	Per cent
Right colectomy	25	3	12.0
Cecectomy	7	1	14.3
Ileocecal resection	3	0	0.0
Colon resections (total)	35	4	11.4
Excision	2	0	0.0
Exteriorization	2	0	0.0
Total	39	4	10.3

diagnosed correctly, in the remainder the diagnosis of malignant disease is often made and this leads to many unnecessary resections of the colon. This error is understandable because of the apparent similarity in the gross appearance of the lesions and the frequent occurrence of a perforating carcinoma. After reviewing the literature, Lauridsen and Ross[5] compiled the results in 153 cases (table I).

The results of the 57 colon resections reported in that series are here given. Thirty-nine patients were operated upon with a mistaken diagnosis of malignancy. The remainder were correctly diagnosed but the inflammatory reaction was such that a resection was necessary. The follow-up findings of the 39 cases

incorrectly diagnosed as carcinoma showed a mortality rate of 10 per cent, as indicated in table II.

The difficulty in diagnosis, even for surgeons of wide experience, is very apparent. Suggestions to be followed during examination of the lesion for evidences of malignancy have included palpation of the area for cauliflower projections into the lumen of the bowel, or invasion of the bowel and ulcerations along the lumen. It has also been suggested that the ostium of the diverticulum may be palpated through the opposite bowel wall, and that frozen sections of lymph nodes or other available tissue should be utilized when possible.

At the Cincinnati General Hospital, 6 cases of diverticulitis of the cecum have been encountered in the past 10 years. These are in addition to those reported from the same institution by Schnug[6] in 1943. A report of these cases is herewith presented.

CASE REPORTS

Case 1. H. S. A 42 year old Negro man was admitted on Oct. 11, 1952. He noted the onset of periumbilical pain 48 hours prior to admission. This pain persisted and shifted to the right lower quadrant on the following day. That evening he became nauseated and vomited once. The following morning, on admission to the Receiving Ward, he had one watery stool. There was no history of a previous similar episode. Physical examination showed moderate tenderness in the right lower quadrant, with guarding and rebound tenderness referred to the right lower quadrant. Total leukocytes numbered 13,500 per cu. mm. The urine was normal. A diagnosis of acute appendicitis was made.

At operation, through a McBurney incision, the appendix appeared normal; but at 2 cm. above the ileocecal valve on the medial side of the cecum there was a rounded, elevated, hard mass measuring 3 cm. in diameter. This was covered with a small amount of fibrin and had a yellowish-gray surface. The hard margins seemed to end fairly sharply in what was thought to be normal cecal wall. A diagnosis of carcinoid of the cecum was made. Through a right rectus incision the right colon was resected and an ileotransverse colostomy was made.

The patient's postoperative course was uneventful and he was discharged on his twelfth postoperative day.

Sections of the gross specimen showed a diverticulum communicating with the lumen of the cecum through a small opening. The cavity of the diverticulum contained a hard fecal mass. Microscopically, the mucosa was necrotic and was densely infiltrated with neutrophiles which extended through the deeper layers as well as the attached adipose tissue.

Case 2. E. H. A 31 year old Negro man was admitted on Oct. 26, 1952. He had developed epigastric pain 48 hours prior to admission. This persisted and then shifted to the right lower quadrant the following day. He continued to eat and to work until six hours prior to admission. At that time, following lunch, his abdominal pain increased and be became anorexic and nauseated although he did not vomit. A laxative taken on the day of onset produced four bowel movements on the day of admission. The patient complained of increased pain on walking and seemed to feel more comfortable with his right leg flexed. There was no history of previous similar episodes. Examination showed the abdomen to be soft throughout, with point tenderness 2 inches above McBurney's point, and with rebound pain referred to this area. Slight tenderness was present in the right flank. Total leukocytes numbered 11,000 per cu. mm. The urine was normal. The diagnosis was acute appendicitis.

At operation, the appendix was found to be normal and was removed. Further search revealed a 3 cm. indurated mass, 3 inches above the ileocecal valve on the anterior medial aspect of the cecum. There was moderate inflammatory reaction in the adjacent bowel, and a fibrous band extending from the mass to the lateral peritoneal wall was severed. The diag-

nosis of acute diverticulitis of the cecum was made. Two Penrose drains were inserted down to the mass and brought out through the McBurney incision, which was partially closed.

The postoperative course was uneventful and the patient was discharged on his twelfth postoperative day. He was followed in the Surgical Clinic, where his wound was noted to be completely healed on the twenty-third postoperative day.

Case 3. R. B. A 38 year old Negro woman was admitted on March 9, 1952. She was well until three days prior to admission, when she noted the onset of lower abdominal *gas* pains. These gradually became cramp-like and shifted to the right lower quadrant and were intermittently sharp. Nausea was present and she vomited three times in two days prior to admission. Daily catharsis and enemas resulted only in diarrheal movements. Some relief was obtained by lying on her side, with her legs drawn up.

This patient had had a total hysterectomy in 1946, at which time the appendix was not removed. Examination showed marked spasm and tenderness over McBurney's point, with rebound tenderness. Bowel sounds were present and high-pitched. Total leukocytes numbered 15,050 per cu. mm. The urine was normal. The preoperative diagnosis was acute appendicitis.

At operation, the appendix was found adherent to an inflammatory mass on the anterior medial surface of the cecum, just above the ileocecal valve. The mass measured 4 cm. in diameter and was rubbery, firm and elevated. The central portion, measuring 1.5 cm., was necrotic but had not perforated. A diagnosis of perforating diverticulitis of the cecum was made. The necrotic area was exteriorized by suturing the parietal peritoneum of the McBurney incision to the cecum around the area of impending perforation. Two days later, a cecostomy tube was inserted into the cecal lumen through this area which had undergone complete necrosis.

The patient did well postoperatively. She was discharged on her twenty-ninth postoperative day, with the cecostomy wound healing satisfactorily. Barium enema showed several diverticula located only in the cecal area. She was followed in the Surgical Clinic until complete healing occurred and has since remained asymptomatic.

Case 4. J. I. A 79 year old white man was admitted Feb. 13, 1951. He stated that he had had a dull epigastric pain associated with fullness and belching, some anorexia, and a weight loss of 40 pounds during the past year. About six weeks prior to admission he suddenly developed severe right lower quadrant pain and noted a mass as large as a baseball in his right lower quadrant. The pain at first was constant and unrelieved by medication. Several days after onset, he passed some bright red blood in his stool; one week later, he passed almost one pint of blood. The patient further reported that the mass gradually decreased in size and that he had become increasingly weaker.

On admission he appeared pale and as if he had been chronically ill. Examination was essentially negative except for slight tenderness and mild increase in muscle tone in the right lower quadrant. His hemoglobin was 11.5 Gm., leukocytes were 8,350 per cu. mm. of blood, and urine was normal. The initial impression was carcinoma of the right colon. A flat plate of the abdomen and a barium enema revealed no disease other than a laminated calculus thought to be in the appendix. These findings seemed to exclude cancer of the right colon and a diagnosis of subacute appendicitis was made.

He was operated upon through a right McBurney incision one week later. The appendix did not appear acutely inflamed and there was no evidence of the previously described calcification. A hard mass was palpable in the cecal wall, just above the ileocecal valve. A diagnosis of cecal diverticultis was made. It was possible to evacuate the contents of the diverticulum into the lumen of the cecum, and the diverticulum was inverted and the adjacent cecal wall approximated with silk sutures. A routine appendectomy was done.

The postoperative course was uneventful and the patient was discharged on his ninth postoperative day. Roentgenograms of the abdomen, taken 48 hours after operation, failed to visualize the previously described laminated calculus. However, a number of calcifications were noted in the descending colon, which were thought to represent the residuals of the mass of calcification which probably was broken up as the contents of the diverticulum

were emptied. The patient showed no recurrence of his previous symptoms but complained intermittently of sour stomach and anorexia. An upper gastrointestinal roentgenographic study revealed no evidence of organic disease six months later.

Case 5. Z. B. A 50 year old Negro woman was admitted on Sept. 1, 1946. The onset of symptoms began 48 hours prior to admission, with sharp right lower quadrant pain, cramp-like in character and occasionally radiating to the right upper quadrant. Nausea was present but vomiting did not occur until the day of admission. These symptoms gradually increased. The patient had had a hysterectomy and appendectomy 25 years prior to this admission. Examination showed an obese woman, with tenderness over McBurney's point, moderate right lower quadrant spasm, and referred rebound tenderness to this area. A sensation of fullness below McBurney's point was described. The total leukocytes were 14,200 per cu. mm. The urine was normal. The diagnosis on admission was neoplasm of the cecum or a possible small bowel obstruction.

At operation, through a right McBurney incision, the cecum was found covered with densely adherent omentum. In freeing the cecum it was found that the appendix was absent, but a hard area was encountered on the posterior lateral surface. On mobilization of this area, a perforation was made into the cecum through extremely friable tissue over the surface of the mass. Tissue for biopsy was taken from the margin of this area and the perforation was closed with Halsted mattress sutures of silk. One Penrose drain was inserted down to the area and brought out through the incision which was partially closed about the drain.

The postoperative diagnosis was diverticulitis of the cecum. The biopsy report several days later showed only chronic inflammation, which was diagnosed as chronic perityphiltis.

The patient's postoperative course was uneventful except for some thin drainage from the wound, and she was discharged on the twentieth postoperative day. The wound was completely healed by the twenty-eighth postoperative day.

Case 6. A. McE. A 72 year old Negro woman was admitted on Sept. 22, 1951. She stated that she had had a mass to the right of the umbilicus for 10 to 15 years, with mild pain over the area. Three weeks prior to admission, she noted the onset of anorexia, weight loss, and an increase in the number of bowel movements per day. One week prior to admission, the pain over the mass became severe and settled in the right lower quadrant. On the day before admission, she had chills and fever followed by several episodes of nausea and vomiting. On admission, examination showed an acutely and apparently chronically ill patient, with a mass 8 cm. in diameter in the right side of the abdomen at the level of the umbilicus, which was described as moving with respiration. There was extreme tenderness over the mass, with rebound tenderness but no rigidity. The total leukocytes measured 21,000 per cu. mm. of blood and the urine was negative. The initial impression was acute cholecystitis, with pericholecystic abscess.

At operation, a hard mass was found in the right colon and cecum, which was fixed to the lateral peritoneal wall. This was diagnosed as carcinoma of the cecum and ascending colon, and a side to side ileotransverse colostomy was performed.

The patient did well postoperatively. Eighteen days later, after preparation of the bowel with sulfasuxidine, a right colectomy was done. The gross specimen showed a mass 4 cm. in diameter on the posterior lateral aspect of the ascending colon, 5 cm. above the ileocecal valve. There was a diverticulum in the mass which communicated with the colon by an 8 mm. ostium. On microscopic sections, this proved to be a true diverticulum although most of the wall was destroyed by inflammation. There was no evidence of carcinoma.

Convalescence was uneventful and the patient was discharged 11 days after the second operation.

COMMENT

These patients present the same problems in diagnosis and treatment which have been encountered in other reports. Appendicitis was the diagnosis in 4 of the 6 cases, and a preoperative diagnosis of diverticulitis of the cecum was not

made in any of them. In 3 of the patients in which the onset of pain was epigastric, periumbilical, or lower abdominal, with later shifting of the pain to the right lower quadrant, the diagnosis of acute appendicitis initially was made. Nausea was present in these 3 patients and vomiting was present in 2. In case 4, the initial diagnosis was carcomina of the right colon; however, this was changed to subacute appendicitis, when a barium enema revealed no disease of the colon and a laminated calculus was found on the roentgenograms in the area of the appendix. Of the remaining 2 patients, 1 was known to have had an appendectomy and was diagnosed as carcinoma of the cecum. The other patient, with a tender mass to the right of the umbilicus, was diagnosed as acute cholecystitis.

At operation, 4 of the 6 cases were correctly diagnosed as diverticulitis of the cecum. Each of these cases was handled differently. In 1 patient—case 2—following appendectomy, drains were placed to the mass and brought out through the incision. The diverticulum was perforated at operation in another patient—case 5—in which an appendectomy previously had been done. Following biopsy section and closure, the area was drained through the working incision. Case 3 showed a necrotic wall of the diverticulum with impending perforation, and this was exteriorized. In case 4 it was possible to evacuate the contents of the diverticulum into the colon after which the diverticulum was inverted and the cecal wall closed. Of the 2 remaining patients who were thought to have malignancy at operation, case 1 was diagnosed as carcinoid of the cecum, and a right colectomy and ileotransverse colostomy were done. Case 6 was diagnosed as carcinoma of the cecum and ascending colon, and a diverting ileotransverse colostomy was made. Later a right colectomy was done. No operative deaths occurred in the 6 patients and the period of hospitalization varied from 9 to 29 days.

The age of the 6 patients varied from 31 years to 79 years, with the average at 52.8 years. Three patients were male and three were female. All but one of the patients were Negroes. Nausea occurred in 5 of the 6 patients, and vomiting occurred in 4 patients.

DISCUSSION

Since diverticulitis of the cecum so closely simulates acute appendicitis from the point of view of onset and symptomatology, appendicitis probably will be mistaken for the correct diagnosis in the majority of patients. As the disease becomes better appreciated, the correctness of the diagnosis at operation should increase. All recommended aids for diagnosis should be fully utilized. The difficulty of distinguishing certain cases from those with perforating carcinoma probably will continue to *tax* surgeons in the future, as it has done in the past.

The choice of operation, when the disease is diagnosed at operation, would seem to vary with the individual lesion. In suitable cases, where the ostium is large and the indurated wall permits evacuation of the contents into the colon, inversion of the diverticulum and closure of the cecum over the base of the diverticulum probably offers the best change of cure, with minimal degree of operative risk or complications. Should the size of the ostium, or the degree of

inflammation, or size of the lesion prohibit this maneuver, closure of the operative wound with or without drains would seem to be the next logical step. When possible, drainage or exteriorization is indicated in patients where perforation or impending perforation is present and inversion cannot be done. Excision of the diverticulum and closure of the cecum, while simple and curative in the majority of patients, is nevertheless attended by a significant although small mortality rate in the recorded cases. Lesions with necrosis and inevitable perforation, which are unsuitable for exteriorization because of position, may lend themselves to excision and closure with drains of the area. Because of the nature and extent of the inflammatory reaction, resection may be indicated in certain patients.

SUMMARY

Six cases of diverticulitis of the cecum occurring at the Cincinnati General Hospital in the past 10 years are presented. The anatomical differences between these cases of *true or congenital* diverticula and the *false or acquired* type, mainly occurring in the sigmoid colon, are presented. Attention is called to the close similarity between this disease and appendicitis and to their usual indistinguishable symptoms and physical findings.

The average age of the patients here discussed was 52.8 years, as compared to the average age of a larger collected series, which was 40.6 years. The disease occurred in three males and three females, which ratio conforms to the usual pattern. Five of the patients had nausea, and 4 had vomiting; this is a much higher incidence than usually is reported for the disease. Preoperatively, 4 cases were diagnosed as acute appendicitis; 1 as carcinoma of the cecum; and 1 as acute cholecystitis. At operation, the correct diagnosis of diverticulitis of the cecum was made in 4 patients. The fifth case was diagnosed as carcinoid of the cecum; and the sixth as carcinoma of the cecum and ascending colon. Each of the last two diagnoses led to bowel resection.

Correct diagnosis of acute diverticulitis of the cecum is nearly always established by the information obtained at operation. Nevertheless, an incorrect diagnosis is made in almost one-third of the cases, which leads to unnecessary colon resections and to a mortality rate of 10.3 per cent; in comparison with 2.2 per cent for those correctly diagnosed. Measures to be utilized in diagnosis at the operating table are discussed.

A plan for surgical management is suggested which should minimize the risk of operative complications.

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STAGING AND PROGNOSIS IN BREAST CARCINOMA

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Notwithstanding its doubtful prognostic value in individual cases, the histologic grading of tumors as first suggested by Broders[1] a generation ago has won almost universal acceptance in the field of surgical tumor pathology. In the field of breast carcinoma, however, the principles of purely histologic grading are difficult to apply with any clinical significance since the vast majority of these tumors show approximately the same degree of dedifferentiation, the major variable being the extent of the associated fibrosis or desmoplasia. Consequently, in the commonly used systems of staging or grading of breast carcinoma, emphasis usually has been placed on gross anatomic features to the exclusion of important histologic and biologic factors influencing prognosis.

FACTORS INFLUENCING PROGNOSIS

In addition to histologic considerations, the following factors are generally considered to have some bearing on the prognosis of breast carcinoma:

1. Axillary metastatic level.
2. Extra-axillary spread.
3. Associated pregnancy or lactation.
4. Subepidermal lymphatic or vascular permeation with or without *inflammatory carcinoma.*
5. Location within the breast.
6. Size of primary tumor.

Histology. Unfortunately, the biologic and clinical behavior of tumors cannot be evaluated on microscopic grounds alone with any degree of certainty, yet there are definite histologic findings in breast carcinoma which merit special consideration from this standpoint.

Noninfiltrating duct or lobular carcinomas—although relatively uncommon—are associated with a more favorable outlook than are infiltrating lesions regardless of type.

Medullary carcinoma with lymphoid stroma—the cellular variety of breast carcinoma showing an associated lymphocytic infiltration of its scanty stroma—as noted by Moore & Foote,[7] carries a considerably better prognosis than does the more common mammary scirrhous variety. In Moore & Foote's series of 52 cases, patients with *medullary carcinoma with lymphoid stroma* had about five times as great a chance for five-year survival as did the scirrhous carcinoma group. This variant of medullary carcinoma then, often regardless of relatively large size, manifests a tendency toward low biologic malignancy and should be treated accordingly in any logical grading scheme.

The same favorable implications should be associated with the papillary variety

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of duct carcinoma[3] and with the diffuse mucoid carcinomas[4], both of which usually show more circumscription and less invasiveness than the average scirrhous carcinoma.

A well preserved acinar pattern in the primary tumor, with glandular or pseudoglandular spaces forming a conspicuous and striking feature of growth, is generally accepted as having favorable prognostic importance. In classifying 154 cases of breast cancer on this basis, Haagensen[4], for example, found the following correlation between five year survival rates and glandular differentiation: marked glandular differentiation—90 per cent five year survival; slight glandular differentiation—36 per cent five year survival; and absent glandular differentiation—24 per cent five year survival. Unfortunately, marked glandular differentiation is a relatively infrequent feature of breast carcinoma, yet it would seem to be a histologic factor worthy of special consideration whenever present.

At the opposite end of the histologic scale are a few rapidly growing anaplastic breast carcinomas, characterized by marked cellular pleomorphism, hyperchromasia and striking mitotic activity, which carry a uniformly poor prognosis; this fact must necessarily be reflected in the final evaluation of these growths.

The fact that the majority of breast carcinomas fall into neither of these categories, but rather into an intermediate group, accounts for the disappointing results of purely histologic grading and creates the need for a more inclusive scheme of staging.

Axillary Metastatic Level. The prognostic importance of axillary metastases has long been emphasized in breast carcinoma with little attention to the *level* of axillary nodes involved by metastatic cancer. Stewart[8] has shown the following correlation between axillary metastatic level and approximate five year survival rate: basal nodes positive—56 per cent survival; basal and central nodes positive—45 per cent survival; apical nodes positive—28 per cent survival. These highly significant findings from the basis for the system of staging in breast carcinoma to be discussed later.

In surgical pathologic practice, the apical segment of the axilla must be studied as a separate specimen which can be arbitrarily defined as that roughly conical portion extending into the medial and apical 5.0 cm. of the axillary fossa, behind and above the pectoralis minor muscle; this includes the apical subclavian group of lymph nodes centered about the thoracoacromial artery. During radical mastectomy, the lower limit of the apical segment should be identified with a long transfixing suture for the orientation of the pathologist. Likewise, in view of their special significance, any separate lymph nodes from the subclavian space or from the region of the thoracoacromial axis stump should—if dissected free—be submitted as apical nodes properly identified.

Extra-axillary Spread. Although the significance of lymphatic extension or metastasis of breast carcinoma to supraclavicular nodes or the internal mammary chain is generally appreciated, the latter group is so seldom examined that its inclusion in a staging system is difficult or questionable. However, on the basis of the studies of Handley and Thackray,[6] it seems reasonable to assume that, given a carcinoma in the medial half of the breast, the presence of axillary

metastases is an indication that invasion of the internal mammary chain has already occurred; this situation must unfavorably influence the final staging of the extent of disease.

As suggested by Handley and Thackray,[5] the internal mammary chain should be biopsied in the second interspace as a routine part of the radical mastectomy procedure. This would not only afford the opportunity of direct pathologic study, but would permit routine ligation of internal mammary vessels and lymphatics in the second and fifth interspaces.

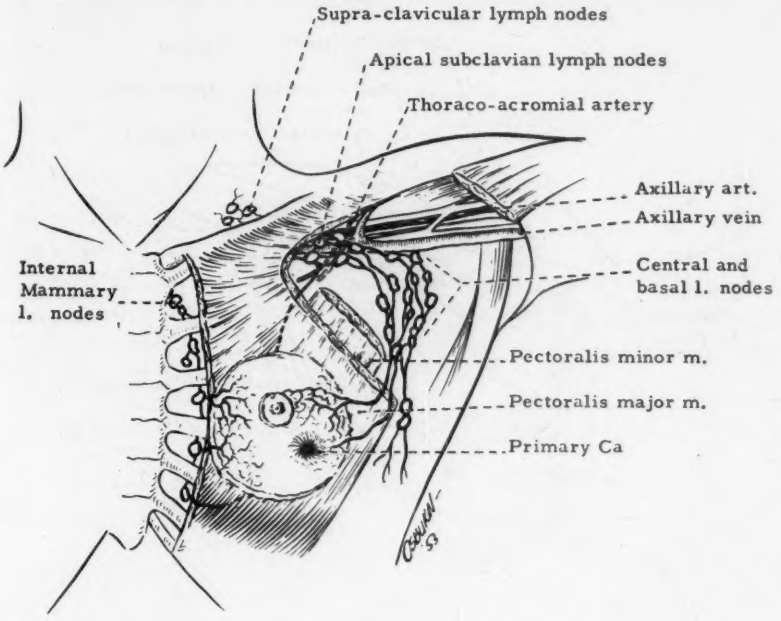
The isolation of the homolateral internal mammary chain by ligation at the upper and lower borders of the breast could be accomplished without significant changes in operative time or mortality in radical mastectomy. Since this lymphatic pathway seems to function as a highly efficient and important route of cancer dissemination and communicates directly with the venous system at its superior extremity, it unquestionably accounts for a high percentage of distant metastases to the pleura, lungs, and liver. If lymphatic extension or embolization could be limited to the internal mammary chain itself, then carcinoma in this area—residual or recurrent after the standard radical mastectomy—would be amenable to treatment by radon implantation or excision by a secondary surgical procedure. Certainly this simple device seems worthy of further study and trial.

Pregnancy or Lactation. It long has been recognized that the prognosis of breast carcinoma is generally unfavorably influenced by coexistent pregnancy or lactation, yet these associated conditions are not invariable criteria of inoperability. At the Memorial Hospital, for example, the five year survival rate for mastectomy cases of this type approximates 25 per cent.[8] Consequently, a few selected cases probably will continue to be subjected to radical mastectomy and pathologic evaluation.

Nonhormonal and purely anatomic factors probably are most important in favoring the dissemination of breast carcinoma during pregnancy or lactation.[9] Increased breast size, rich lymphatic development, and parenchymal hyperplasia with relative reduction of fibrous stroma are the most significant of such factors and probably account for the predisposition to the development of *inflammatory* signs in breast carcinoma during pregnancy and lactation. In the group reported by Meltzer,[9] approximately 25 per cent of breast carcinomas associated with pregnancy and 40 per cent of those associated with lactation were of the *inflammatory* type.

Subepidermal Lymphatic or Vascular Permeation. Permeation of dermal lymphatics or vascular spaces by carcinoma associated with cutaneous lymphedema and passive hyperemia is, of course, the pathologic basis for the relatively uncommon clinical entity of *inflammatory carcinoma* of the breast. The anticipated poor prognosis in this type of case is borne out by the reported average survival time of two years in the group studied by Taylor and Meltzer.[9] Consequently, whenever present, microscopic or clinical evidence of cutaneous lymphatic extension—like associated pregnancy or lactation—should be incorporated in the realistic staging of breast cancer as important factors unfavorably influencing prognosis.

Location in Breast. Medial half breast carcinomas have been considered to have a less favorable group prognosis because of their tendency toward early intrathoracic lymphatic extension via the internal mammary chain. This concept is supported by the work of Handley and Thackray[6] who demonstrated that with medial half breast cancers the internal mammary chain is involved at the



STAGE I

PRIMARY CARCINOMA LIMITED TO BREAST

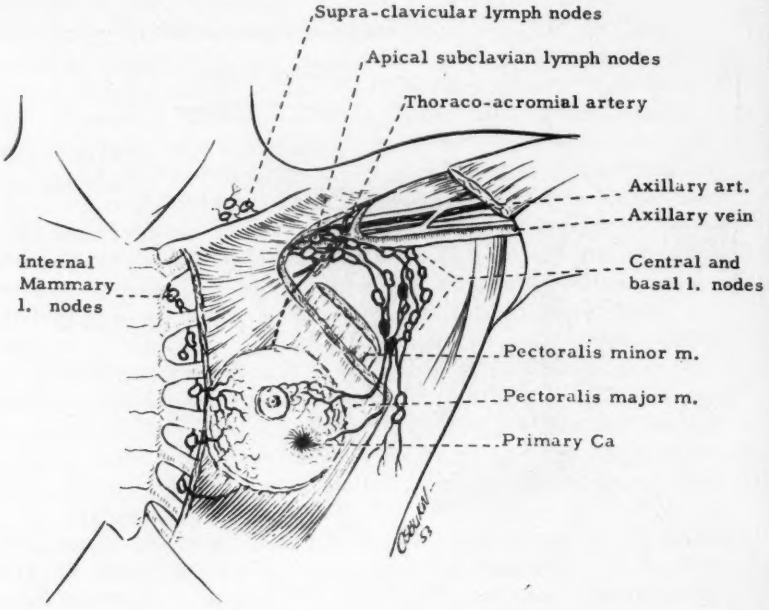
EXPECTED FIVE YEAR SURVIVAL RATE - APPROXIMATELY 85%

FIG. 1. Stage I. Primary carcinoma limited to breast—expected five year survival rate—approximately 85 per cent.

same time as—or before—the axillary lymph nodes are invaded. Consequently, it would seem reasonable to consider this finding as a significant factor unfavorably influencing prognosis whenever present.

Size of Primary Lesion. Perhaps the best established and most reliable index of relative group prognosis in breast carcinoma is the size of the primary tumor, as noted by the examining pathologist. In the Memorial Hospital series of approximately 1400 breast carcinomas cited by Stewart[8], a striking correlation was noted between tumor size and five year survival rates. Patients with primary tumors less than 2.0 cm. in diameter had five year survival rates of approximately

90 per cent to 50 per cent depending on whether or not axillary involvement was demonstrable. With primary lesions of 4 to 5 cm. in diameter however, the comparable survival rates dropped to 60 per cent and 30 per cent. These findings suggest that primary scirrhous carcinomas which exceed an arbitrary mean size should be considered unfavorable from a prognostic standpoint, and should be evaluated accordingly in the final staging of the tumor.



STAGE II

METASTASIS TO CENTRAL OR BASAL AXILLA ONLY

EXPECTED FIVE YEAR SURVIVAL RATE - APPROXIMATELY 50%

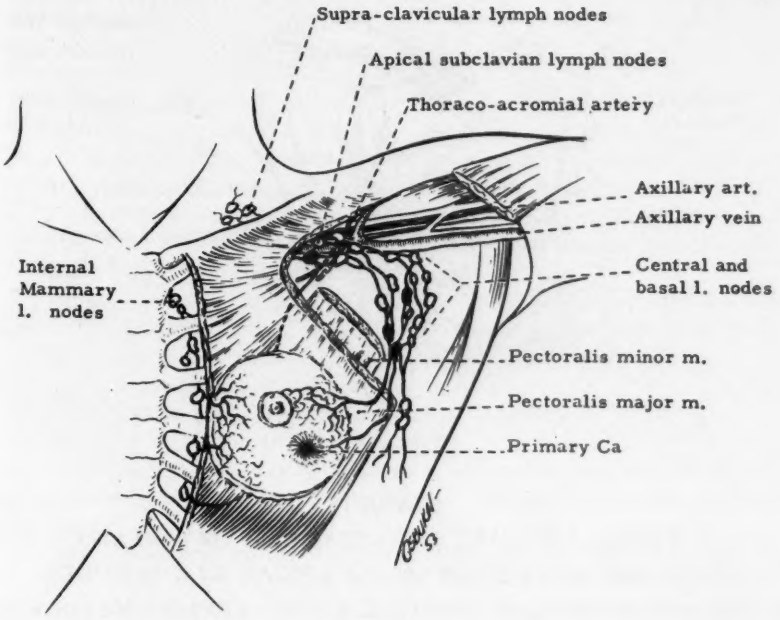
FIG. 2. Stage II. Metastasis to central or basal axilla only—expected five year survival rate—approximately 50 per cent.

PROPOSED STAGING OF BREAST CARCINOMA

It would be helpful from the standpoint of group follow-up and surgical classification, if the various clinical, anatomic, histologic, and statistical factors outlined above could be integrated into a simple scheme which would permit realistic staging of breast carcinoma. Obviously, too many hidden variables are operative in the individual case for such a system to have individual prognostic value. Yet, for purposes of therapeutic evaluation studies and crude group prognosis, such a system of staging would seem to have some merit.

Using an anatomic grouping, the following basic stages of infiltrating breast carcinoma are proposed:

Stage I. Primary lesion limited to breast without demonstrable extramammary extension by routine pathologic study. Expected five year survival rate 85 per cent (fig. 1).*



STAGE III

METASTASIS TO APICAL AXILLA

INVOLVING 50% OR LESS OF APICAL LYMPH NODES

EXPECTED FIVE YEAR SURVIVAL RATE - APPROXIMATELY 30%

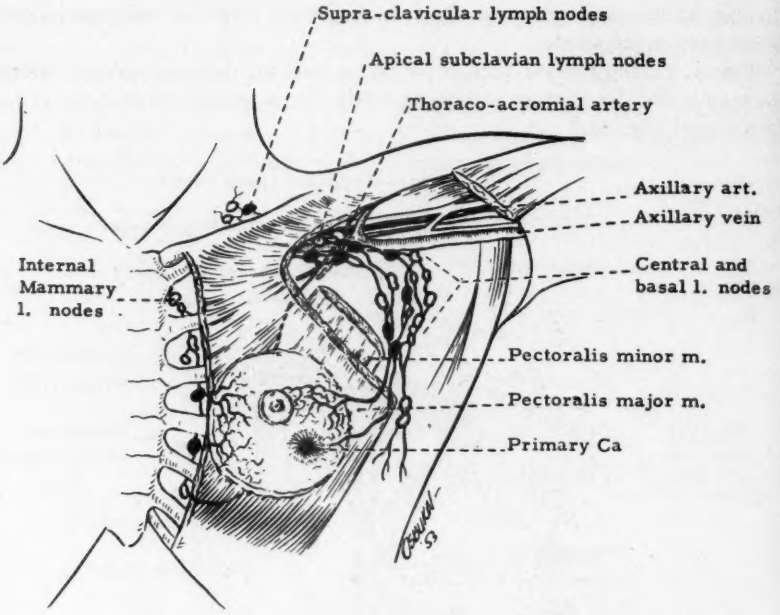
FIG. 3. Stage III. Metastasis to apical axilla—involving 50 per cent or less of apical lymph nodes. Expected five year survival rate—approximately 30 per cent.

Stage II. Primary lesion with demonstrable metastasis in central or basal axillary nodes only. Expected five year survival rate 50 per cent (fig. 2).

Stage III. Primary lesion with apical lymph nodal metastasis involving 50 per cent or less of demonstrable apical axillary nodes. Expected five year survival rate 30 per cent (fig. 3).

Stage IV. Primary lesion with extra-axillary metastasis *or* metastatic involve-

* The line drawings were prepared by Mr. William A. Osburn, whose assistance is gratefully acknowledged.



STAGE IV

EXTRA-AXILLARY METASTASIS OR METASTASIS INVOLVING MORE THAN 50% OF APICAL LYMPH NODES

EXPECTED FIVE YEAR SURVIVAL RATE - APPROXIMATELY 15%

FIG. 4. Stage IV. Extra axillary metasis or metastasis involving more than 50 per cent of apical lymph nodes. Expected five-year survival rate—approximately 15 per cent.

TABLE I

Factors influencing prognosis in breast carcinoma

A. *Unfavorable—increase basic stage by I:*
   1. Scirrhous primary over 3.0 cm. in diameter.
   2. Coexistent pregnancy or lactation.
   3. *Inflammatory* signs or vascular or lymphatic permeation.
   4. Medial half lesion with positive axillary lymph nodes.
   5. Anaplastic rapidly growing carcinoma.

B. *Favorable—decrease basic stage by I:*
   1. Medullary carcinoma with lymphoid stroma.
   2. Infiltrating papillary duct carcinoma.
   3. Marked preservation of glandular or pseudo-acinar pattern.
   4. Diffuse or extensive mucoid degeneration.

ment of more than 50 per cent of demonstrable apical axillary lymph nodes. Expected five year survival rate 15 per cent (fig. 4). Although often uncertain or indeterminate preoperatively, this stage is obviously inoperable, since Da-

land's[2] series of 100 consecutive *untreated* cases showed a better five year survival rate of 22 per cent.

Noninfiltrating breast carcinomas of the intralobular or intraductile type, including the noninfiltrating intraductile papillary and comedocarcinomas, are not considered, since they constitute only about 1 per cent of breast carcinomas and should carry a uniformly good prognosis when treated by simple mastectomy.

Having established the basic stage as noted above, this may be shifted one stage only in an *unfavorable direction* by the presence of one or more of the following factors (table I):

1. Scirrhous primary carcinoma measuring over 3.0 cm. in diameter.
2. Coexistent pregnancy or lactation.
3. *Inflammatory* signs (pigskin lymphedema, redness and heat, clinically) or lymphatic or vascular permeation on microscopic examination.
4. Medial half breast carcinoma with positive axillary lymph nodes.
5. Anaplastic carcinoma with marked cellular pleomorphism and hyperchromasia with striking mitotic activity.

In the same manner the basic stage may be shifted one stage only in a *favorable direction* by the finding of one of the following histologic types of carcinoma:

1. Medullary carcinoma with lymphoid stroma.
2. Infiltrating papillary duct carcinoma.
3. Striking or marked preservation of acinar or pseudo-acinar pattern.
4. Diffuse or extensive mucoid degeneration.

In the event that favorable or unfavorable factors are not applicable or *cancel out*, the original anatomic stage is retained as the final stage for classification purposes.

It should be emphasized that the expected five year survival rates, associated with the various stages, are simply crude approximations based on previous studies[8] and are in no sense observed rates based on personal experience or new data.

SUMMARY

Factors influencing group prognosis in breast carcinoma are briefly considered and a scheme of staging is proposed which attempts to integrate histologic, gross pathologic, and clinical findings to produce a more realistic final staging, useful in follow-up and therapeutic evaluation studies.

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THE EFFECTS OF ANTIHISTAMINE DRUGS ON FIBROPLASIA

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In previous articles, one of us (J. K. B.) described the effects of fibroblastic agents in the production of perivascular fibrosis.[1, 2] The present study was undertaken to evaluate various substances which might be used to delimit fibroplasia. Injuries were classified into two large groups: (1) those inflicted with minimal destruction and irritation of cells resulting in restricted inflammation, exudation and granulation and healing by what is commonly known as first intention (per primam intentionem); and (2) wounds produced by widespread destruction or with continued irritation of cells resulting in severe inflammation, prodigious quantities of granulation tissue and provoking repair by what is commonly known as healing by second intention (per secondam intentionem). It, of course, follows that wounds in group 1 will heal with minimal scar and wounds in group 2 will heal with various degrees of maximal scar or cicatrization.

THE USE OF ANTIHISTAMINES

We have been unable to find mention of the use of the antihistamine drugs as fibroblastic inhibitors. Their possible use was first suggested in experiments which were designed to study the effects of histadyl,* on hypertension which was experimentally produced in dogs by wrapping their kidneys in a silk envelope dusted with sulfathiazole powder. The animals failed to develop hypertension and the cause was not revealed until re-exploration or postmortem examination disclosed that very little scarring had occurred. This finding was the reverse of previous experiences with the use of this method for the production of an ischemic kidney. These observations prompted us to use histadyl as well as other agents in an attempt to delimit fibroplasia induced by chemical or mechanical irritations in the peritoneal and pleural cavities.

EXPERIMENTAL PROCEDURES

Group 1. Effect of histadyl on fibroplasia induced by silk envelopes dusted with sulfathiazole powder.

Two dogs were used as controls. Dog ZH 43, blood pressure 125/80; weight 12 kilograms, was anesthetized with sodium pentobarbital, 30 milligrams per kilogram of body weight, given intraperitoneally. Through bilateral lumbar incisions both kidneys were wrapped in dry silk envelopes under which had been placed sulfathiazole powder. Four months after operation, the blood pres-

From the Departments of Surgery, Indianapolis General Hospital and the Indiana University Medical Center. This study was aided by a grant from the Charles J. Wolf Foundation for Medical Research. We are greatly indebted to the staff of the Lilly Research Laboratories for many helpful suggestions and for the generous supply of drugs used in this work.

* Histadyl—Thenylpyramine—Hydrochloride of N,N-dimethyl-N′(2-thenyl)-N,-(2-pyridyl)-ethylenediamine (Lilly).

sure was 230, pulse 100 and blood urea nitrogen 11.8 milligrams. This dog died after eight months and postmortem examination showed an enormous amount of fibroplasia around both kidneys superficially under the intact silk envelope (fig. 1A). This scar tissue involved the lower lobes of the liver, pancreas and adrenals and was about 5 by 6½ inches in size. This we consider to be a typical fibroblastic response to the envelopment of a normal kidney by silk in which sulfathiazole powder has been dusted. Dog ZH 40 was treated in exactly the same

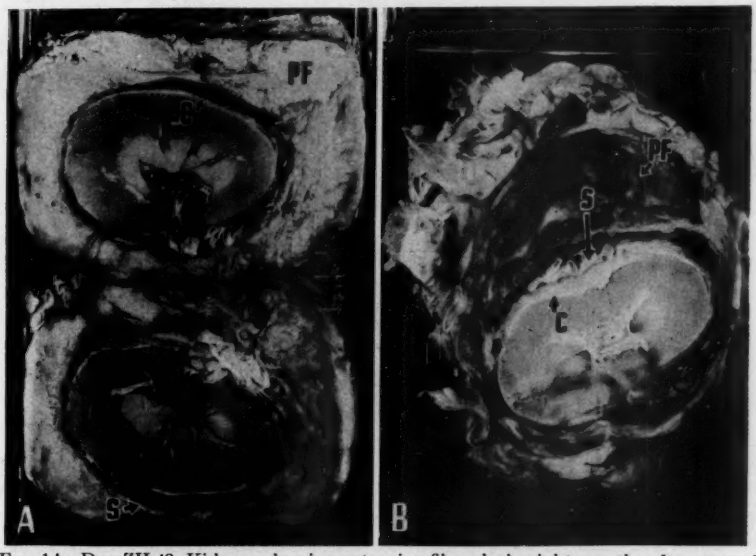

FIG. 1A. Dog ZH 43. Kidneys showing extensive fibroplasia eight months after wrapping in a dry silk envelope which had been dusted with sulfathiazole powder.
(C) Capsule
(PF) Pericapsular fibroplasia
(S) Silk envelope

FIG. 1B. Dog ZH 40. Postmortem photograph of the left kidney which had been encased in a silk envelope dusted with sulfathiazole powder for eight months.
(C) Capsule
(PF) Pericapsular fibroplasia
(S) Silk envelope

way. The same studies were made and the blood pressure rose to 230 millimeters of mercury within seven months. Postmortem findings eight months after surgery showed the left kidney to be encased in a huge mass of scar about 8 inches in diameter. The spleen and pancreas were also involved in scar tissue (fig. 1B).

Three dogs were treated with histadyl. Dog 52B4 weighed approximately 13 kilograms and his blood pressure varied from 145 to 120 systolic and from 80 to 88 diastolic. On June 29, 1949, he was started on 200 milligrams of histadyl orally each day. On July 1, he was anesthetized with intraperitoneal sodium pentobarbital, 30 milligrams per kilogram of body weight. A right nephrectomy was done through a right lumbar incision; and through a left lumbar incision the

left kidney was wrapped in a heavy silk envelope which had been dusted with sulfathiazole powder. On July 8 the dog's blood pressure was 120/100 and the histadyl seemed to be causing some vomiting. Therefore, the dose was reduced to 100 milligrams given twice a day. From July 11 to September 14 the blood pressure varied between 125/80 to 155/72 and the blood urea nitrogen remained normal, ranging from 33 to 47 milligrams per cent. On Oct. 29, 1949, the dog was again anesthetized and a left nephrectomy was done. There was almost no fibroplasia around the kidney although it had been wrapped for four months (fig. 2A).

Dog 52B19 was treated, as was the previous animal, on Sept. 24, 1949. The histadyl, which was started on September 12, 200 milligrams daily, was con-

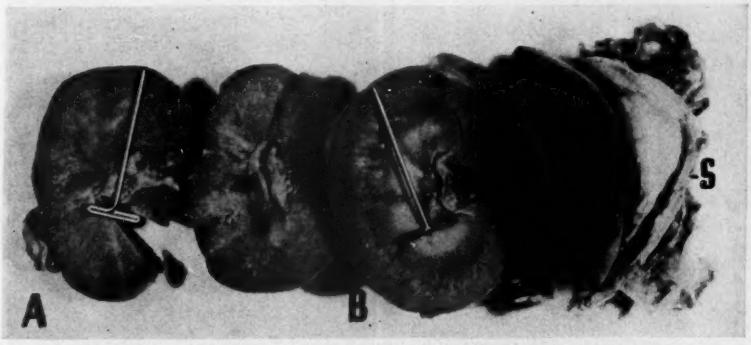

FIG. 2A. Dog 52 B4. Left kidney showing little fibroplasia four months after wrapping with silk envelope which had been dusted with sulfathiazole powder. This dog received histadyl before and after wrapping.

FIG. 2B. Dog 52B19. Left kidney 23 months after wrapping with silk envelope which had been dusted with sulfathiazole powder. This dog also received histadyl before and after operation. Note the minimal fibroplasia.

(S) Silk envelope

tinued postoperatively but no hypertension developed. On Aug. 14, 1951, the dog died. Postmortem findings showed the left kidney with minimal fibroplasia 23 months after operation (fig. 2B).

Dog 52B11 was started on histadyl Sept. 5, 1949. Three days later he was anesthetized and treated in exactly the same way as the 2 previous animals were treated. From September 8 until November 10, the blood pressure varied between 125/80 and 180/100, but there was no progressive increase in pressure. On Sept. 21, 1950 a Goldblatt clamp was placed about the left renal artery and four days later the dog died. Postmortem examination showed practically no fibroplasia around the left kidney although it had been wrapped for 12 months (figs. 3A and 3B).

Group 2. Effect of histadyl on fibroplasia induced in pleural and peritoneal cavities by irritants and trauma.

Six dogs were used as controls. Two-tenths (0.2) grams of talc per kilogram of body weight were placed within the pleural cavity and 1 gram of talc per

kilogram of body weight was deposited in the peritoneal cavity in definitely demarcated areas. In the chest it was introduced over the diaphragm and in the costophrenic angle. In the abdomen it was placed in one of the lower quadrants. Two months later the animals were reexplored and the extent of adhesions was carefully noted. The controls invariably showed extensive reaction. The pleural

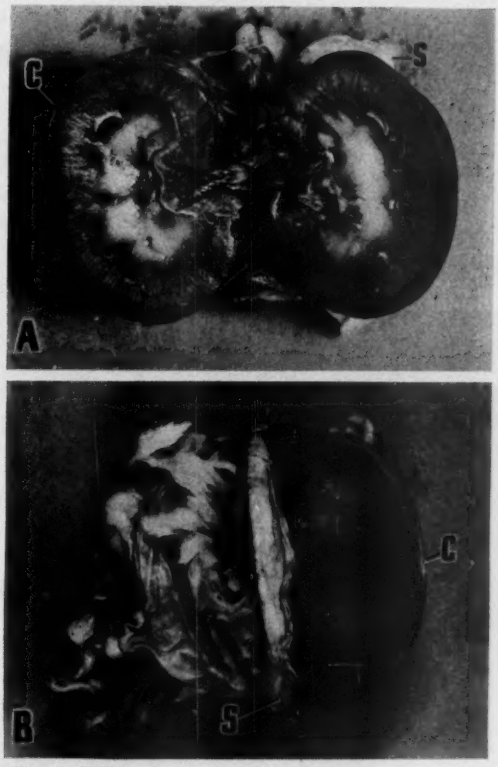

FIG. 3A. Dog 52B11. Left kidney one year after wrapping with a silk envelope which had been dusted with sulfathiazole powder.
(C) Capsule
(S) Silk envelope
FIG. 3B. Lateral view showing the envelope after it had been stripped from the capsule. There is complete absence of fibroplasia.

cavity contained 200 to 300 cc. of fluid with dense adhesions, and the intestines were hopelessly matted together throughout the area on which the talc had been placed. Two dogs were treated by denuding 12 inches of intestine with rough gauze, then placing 2 grams of talc on the denuded area. This causes a severe reaction with dense adhesions after 60 days.

Six dogs were treated with histadyl—exactly the same quantity of talc was introduced into approximately the same areas in the pleural and peritoneal

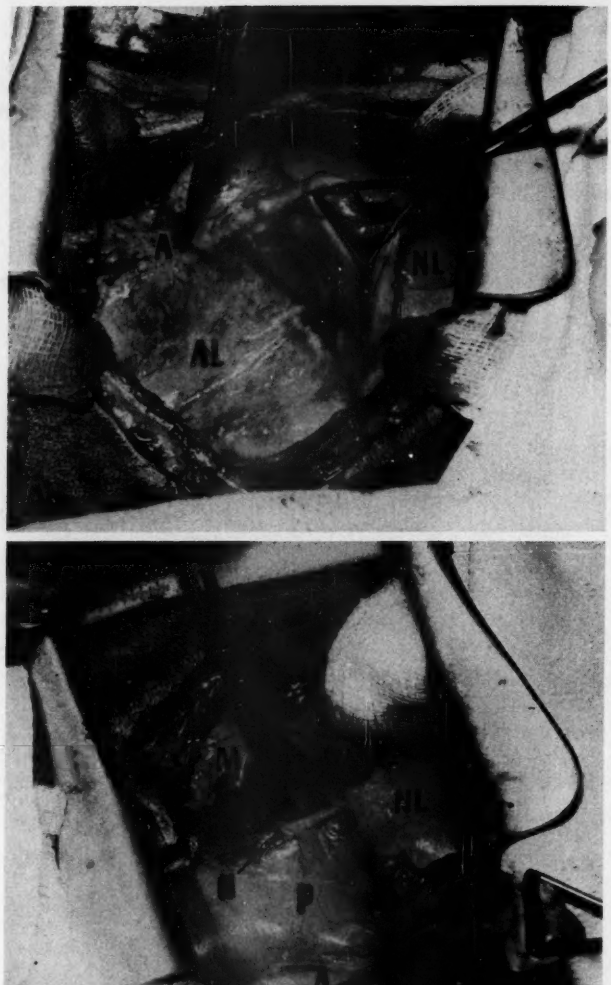

FIG. 4A. Dog B217. Photograph taken two months after lower lobectomy and installation of 2.6 grams of sterile talc (0.2 grams per kilogram of body weight) in the left pleural cavity. There are dense adhesions between the lung and parietal pleura, mediastinal pleura and diaphragm.

(A) Adhesions between lobes
(AL) Lower lobe adherent to the pericardium
(NL) Normal lung

FIG. 4B. Dog B216. Photograph of left pleural cavity eight weeks after installation of 3 grams of sterile talc (0.2 grams per kilogram of body weight) following lower lobectomy. This dog received 50 milligrams of histadyl intramuscularly daily for four weeks.

(M) Mediastinal pleura
(N) Phrenic nerve
(P) Pericardium
(AO) Aorta
(NL) Normal lung
(A) Adhesion

Note the paucity of adhesions as compared with 4A.

cavities. However, in addition, these dogs were given 200 milligrams of histadyl daily by mouth and in 1 of the dogs 50 milligrams of histadyl were given intramuscularly each day. Eight weeks later the histadyl dogs were operated upon and a striking paucity in the number of adhesions was noted.

Group 3. Effect of histadyl on fibroplasia, the result of standard thoracic and abdominal surgical procedures with the introduction of irritants.

Four dogs were used as controls. The animals were prepared as in group 2 and the same experiments were made with the exception that in the pleural cavity lobectomy was done and in the abdomen a partial gastric resection with restoration of continuity by the anterior polya method was done. It was noted that the control dogs in which talc had been placed two months previously had severe reactions (fig. 4A). One of the dogs died of peritonitis, and 1 in which the talc had been introduced into the pleural cavity developed an empyema and ultimately died from it. In every dog, after two months, the peritoneal cavity was filled with dense adhesions not only in the area in which it had been introduced but also surrounding the operative sites.

The same experiments were done in 4 dogs treated with histadyl. The animals with operations in the pleural cavity were given 200 milligrams of histadyl daily by mouth. Those that had partial gastric resections were given 50 milligrams daily intramuscularly until they could swallow the drug by mouth. The dogs survived the operative procedures and were observed for two months at which time the pleural and peritoneal cavities were reexplored. Reactions were minimal (fig. 4B).

Group 4. Effects of histadyl on fibroplasia in the peritoneal cavities of small animals. Fourteen guinea pigs and 48 rats were used.

The animals were anesthetized with .09 of a milligram of pentobarbital sodium intraperitoneally for each gram of body weight. This dose was found to be too large because five of the guinea pigs died shortly after operation and therefore the quantity was reduced to .085 milligrams. A longitudinal incision was made in the abdominal wall and a loop of bowel either small or large was marked with a simple suture of no. 00000 silk on the antimesenteric border. Sterile talc was stroked on either side of the suture for a distance of approximately 1 to 1½ centimeters. The loop of bowel was replaced into the peritoneal cavity and the incision was closed. Half of the animals were used as controls and half were given daily doses of 4 milligrams of histadyl subcutaneously. This represents approximately three-fourths of the lethal dose and was continued for a period of two weeks. They were reexplored four weeks later.

Results: The histadyl group of guinea pigs and rats showed minimal adhesions on postmortem findings and there was very little foreign body reaction on microscopic examination (figs. 5A and 5B). However, in the control group foreign body reactions were severe with the exception of 1 guinea pig that died three days after operation due to evisceration (figs. 6A and 6B).

Another set of controls was run in an effort to evaluate the degree of fibroplasia by simply opening the abdomen with or without histadyl. This was done on 4 of the guinea pigs. There was no difference in the two groups.

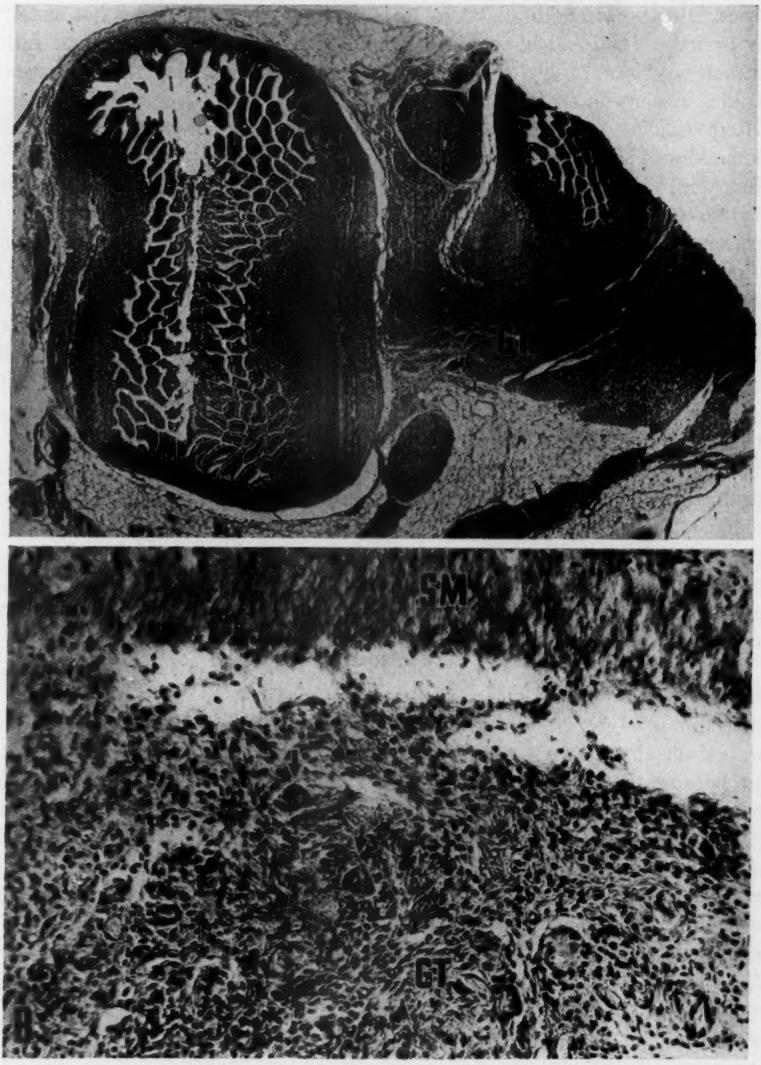

FIG. 5A. Intestines from a rat in which sterile talc had been placed on a loop of intestine over a distance of 1 to 1½ centimeters four weeks previously. Note the enormous amount of fibroplasia between the adjacent loops.

(GT) Granulation tissue; the rectangle shows the area from which the microsection shown in 5B was taken.

FIG. 5B.

(SM) Smooth muscle of intestine

(GT) Granulation tissue. The arrows point to foreign body giant cells.

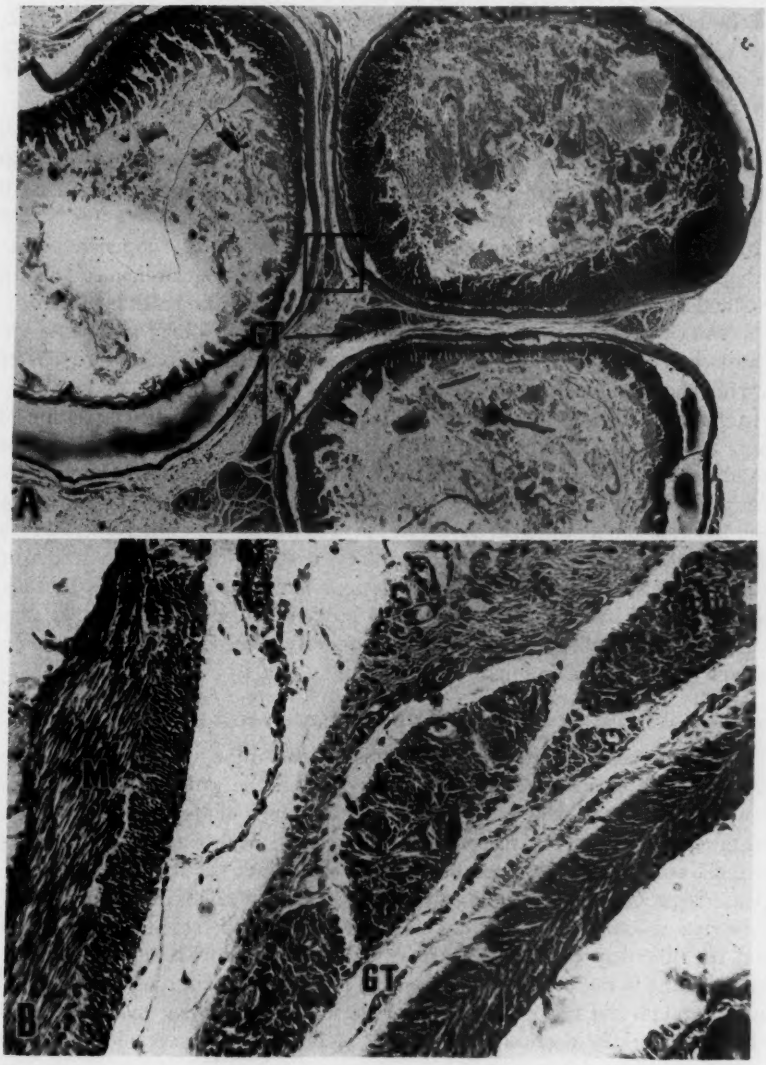

FIG. 6A. Adjacent loops of bowel from a rat in which sterile talc had been deposited four weeks previously. This animal received 4 milligrams of histadyl subcutaneously each day.
(GT) Granulation tissue is minimal as compared with 5A. The square indicates the area from which the microsection shown in 6B was taken.

FIG. 6B. Note the small amount of granulation tissue as compared with figure 5B.
(M) Muscle layer
(GT) Granulation tissue. The arrows point to foreign body giant cells.

This indicates that histadyl is probably effective only where continuous irritation has been used to stimulate fibroplasia. Histadyl did not in any way affect the degree of fibroplasia in the incision in any of the animals.

DISCUSSION

Wounds will not heal without an inflammatory response; because without inflammation there could not be the sequence of events which leads to the formation of granulation tissue.

Inflammation has been thought to be initiated by one of two mechanisms or perhaps both: Krogh[5] and Lewis[6] were of the opinion that mechanical, thermal or chemical stimuli cause the release of an histamine-like substance which, in turn, causes the local vasodilatation. Local axonal reflexes may also cause vasodilatation, and it is highly probable that the nervous impulses are effective through chemical mediators.

In contradistinction to an acute injury, a chronic or continued irritation produces a somewhat different reaction because of the H-substance and probably the liberation of a less diffusible agent which has been called an H-colloid substance by Krogh[5] or a *slow acting substance* by Feldberg.[3] Other compounds have been thought to play a role in the initiation of this response; namely, certain polypeptids which may be extracted from inflammatory exudates and which may cause the release of histamine or histamine-like substances; also, McIntyre[8] observed that histamine can be liberated in vitro and in vivo by simple amines or more complex alkaloids. Perhaps histamine is released through a proteolytic enzyme system, involving many mechanisms.

CONTROL OF INFLAMMATION

Inhibitors such as antifibrinolysin normally control the inflammatory response. Antihistamine drugs may be used to accomplish the same purpose in experimental animals and Haley and Harris[4] have shown that the drugs will block the mammalian capillary response to histamine when topically applied. When a minimal amount of tissue cell damage has been induced the inflammatory response is probably more the result of a local axonal reflex than of chemical or endocrine action. The reason for this is not clearly understood but it is possible that the difference lies in the intensity of the stimulus with more exudation induced by the more severe types of injury. This may explain the observation that antihistamines did not prevent fibroplasia in wound healing by first intention, but did materially decrease the amount of fibroplasia in wounds subjected to severe or continued irritation.

Our experiments have not included a study of the effects of hyaluronic acid and hyaluronidase. However, Mayer[7] has demonstrated that the hyaluronidase content in inflammatory tissue is much greater than in normal tissues. Moreover the hyaluronidase spreading factor can be almost completely controlled by antihistamine drugs.

Antihistamines may be helpful in delimiting fibroplasia in some clinical states. Keloids, in which the irritating factor is not definitely known, could conceivably

be controlled. Excessive scarring might be prevented following such operative procedures as pneumonolysis, pericardiectomy for constrictive pericarditis and division of extensive abdominal adhesions to relieve bowel obstruction.

We have not determined the relative effectiveness of various antihistamine drugs; the length of time that they should be given postoperatively; nor the maintenance dose. However, we believe that the drug should be continued in small doses for a minimum of six months after operation so that the irritant may be completely localized.

CONCLUSIONS

1. Antihistamine drugs definitely limit the amount of fibroplasia in experimental wounds produced by severe or continued trauma. This was not observed in clean incisions with minimal damage to cells.

2. Possible clinical uses are suggested.

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ACUTE OBSTRUCTION OF THE LEFT COLON

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In complete colonic obstruction, the site of the obstruction lies within the left half of the colon in approximately 85 per cent of cases; Burgess[2] reports a figure of 87.6 per cent and Gregg and Dixon[6] in a similar series report the point of obstruction distal to the hepatic flexure in 84.7 per cent. Primary carcinoma of the bowel overwhelmingly is the most common cause of such obstruction.

There is virtual unanimity of opinion that the acutely obstructed colon constitutes a grave emergency requiring prompt surgical intervention and an almost equal measure of agreement that such intervention should not take the form of a direct attack on the obstructing lesion either by resection and primary anastamosis or by any type of exteriorization procedure, but that some type of preliminary decompressive procedure should be done.

The factor which lends particular urgency to the need for decompression is based on the fact that most obstructions of the left colon are of the *closed loop* variety owing to the competency of the ileocecal valve. Sperling[11] has shown competence of the ileocecal valve in the dog with intraluminal pressures up to 50 cm. of water, and that the bowel wall subjected to such pressures develops gangrene and necrosis. Intraluminal pressures in this range were found in colonic obstruction in human beings. Dennis[4] estimates that competency of the ileocecal valve is present in 61 per cent of cases of obstruction, but states that perforation of the cecum may occur even with an incompetent ileocecal valve and mentions a case previously reported by Rigler in which perforation of the cecum occurred in the presence of marked ileac distention and a presumably incompetent ileocecal valve. It is our opinion, however, that such distention of the distal ileum cannot be relied upon as evidence of incompetency of the ileocecal valve, but that in many cases of advanced obstruction, the greatly distended cecum no longer will accept any further increment of small bowel content and that there then exists what amounts to an obstruction of the small bowel at the ileocecal valve superimposed on a closed loop obstruction of the colon.

The presence of distended loops of small bowel, therefore, should not be construed as eliminating the danger of cecal necrosis and perforation nor lead one into the error of prolonged trial of suction drainage with the Miller-Abbott tube in the vain hope that obstruction of the colon can be relieved thereby. Michel[9] reports that when the Miller-Abbott tube intubation was used as a first therapeutic step, 15 of 34 patients died. Of 10 of these patients who improved temporarily and operation was withheld, 6 or 60 per cent died. The use of such tubes with suction is a valuable adjunct in treatment and should be routinely used as such, but surgical intervention should be delayed not longer than is

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necessary to restore the patient's fluid and chemical balance, hematocrit and blood volume to normal levels.

With regard to the type of decompressive procedure which should be used in the patient with acute obstruction of the left colon, there is much less uniformity of opinion among the writers on this subject. For many years cecostomy has been the procedure of choice of most surgeons. Haggard[7], Graham[5], Rankin and Johnston[10] and Allen and Welch[1] have favored cecostomy, although Rankin and Johnston state that when possible it is advisable to exteriorize the cecum or a portion of the ascending colon. Many of the proponents of tube cecostomy admit its limitations, but favor it because of its supposed simplicity.

Advocates of tube cecostomy, as pointed out by Cheever[3], have contended that it is easier to make; that it gives sufficient escape of colonic contents to relieve the obstruction and to permit adequate cleansing of the bowel preparatory to resection; that in most cases it will close spontaneously. As Cheever also notes, all but the last of these statements are frequently untrue and he argues forcefully in favor of complete diversion of the fecal stream. Judd[8], Wangensteen[12], Dennis[4] and Michael[9] have all expressed preference for the principle of complete diversion of the fecal stream.

As our own experience has developed, we have found that our initial preference for cecostomy has changed to a firm conviction that transverse colostomy—when done according to principles that will be elaborated below—is infinitely superior as a decompressive procedure in acute obstruction of the left colon.

The advantages of transverse colostomy are manifold as compared with tube cecostomy. First, the right half of the colon, which is often greatly distended with thick, putty-like feces, empties by its own peristaltic action when a vent is provided; the vent provided is the full diameter of the lumen of the bowel after the first 24 hours; the bowel wall of the transverse colon is relatively healthy compared with the thinned-out, soggy cecal wall, and the usual presence of a long mesentery facilitates the procedure; the danger of the tube being pulled out by an irrational patient with possible fatal outcome is eliminated; the decompression is done aseptically after the bowel has been exteriorized and the wound closed and walled off by heavy vaseline gauze dressings; the colon is completely defunctioned. Through and through irrigations prepare the bowel for subsequent resection; infection of the colostomy wound rarely occurs; the opening is removed from the area of subsequent incision for resection. Last, the simplicity of the procedure recommends it to us and in our opinion, it is a simpler procedure than tube cecostomy.

In contrast with the above, in our experience with tube cecostomy, decompression often fails completely, and is unsatisfactory in many more cases. Retrograde washing out of the colonic contents by irrigating through the tube is cumbersome and unsatisfactory and when the contents of the right colon consist of a thick mass of feces, it is virtually impossible. The cecal wall is so sodden, thinned-out, and distended in many cases, that an aseptic cecostomy is a virtual impossibility and infection of the wound is common. The omentum is often out of reach

and difficult to bring down to interpose between the cecal wall and the parietal peritoneum. Last, the degree to which it facilitates and makes safe the subsequent resection falls far short of that accomplished by transverse colostomy. The one obvious advantage of cecostomy is that in most instances, except with the Graham[5] type of cecostomy, it will close spontaneously.

In the execution of any decompressive procedure in a patient with an acutely obstructed bowel, the immediate objective should be effective decompression and the operation should be simple; should be done quickly with minimum disturbance of the patient. Attempts at exploration at this time increase greatly the morbidity and frequently lead to fatality and should not be attempted. In doing a transverse colostomy, these facts must be kept constantly in mind. As emphasized by Wangensteen[12] and Dennis[4] a transverse incision should always be used; first, because it is parallel to the long axis of the transverse colon and, second, and equally important in a greatly distended patient, it does not gape; and if the incision is kept short, as it should be, the surgeon is not hampered by a mass of small intestine being extruded into the operative field. The procedure, as we have used it, is simple in the extreme and consists of a short transverse skin incision to the right of the midline, 2.5 to 3.5 inches in length—depending on the thickness of the abdominal wall—carried down through the anterior rectus sheath over the lateral border of the rectus muscle width division of the lateral 1 to 1.5 inches of the muscle fibers, and the peritoneum. The great omentum is found easily and is picked up and drawn upwards until the lower margin can be grasped. Gentle traction is then exerted on the omentum until the colon comes into view. The increased intra-abdominal pressure actually facilitates this maneuver. The distended colon is then slowly brought up into the incision until it can be grasped. A blunt nosed clamp is thrust through the mesentery and a thick glass rod covered with rubber tubing is passed through. If the opening in the peritoneum and anterior rectus sheaths have been gauged properly, no closure is necessary beyond one or two silk stitches in the skin. Occasionally, one or two interrupted sutures may have to be placed in the peritoneum or fascia. In rare instances, when one encounters adhesions from previous surgery, it is necessary to enlarge the incision. No exploration, except for sweeping a finger over the right lobe of liver, is done. A thick vaseline gauze dressing is placed around the exteriorized loop and a no. 28 catheter is inserted and fixed with a purse-string suture. If the distention of the cecum is great, the catheter can be directed into the ascending colon by placing the index finger of the left hand in the peritoneal cavity beneath the dressing and using it to guide the tube as it is passed through the hepatic flexure.

The anterior wall of the exteriorized loop is divided at right angles to the long axis of the bowel in 24 or 48 hours, depending on the urgency of the particular case. The posterior wall is left intact in order to hold the loop up over the glass rod which is left in place for 10 to 14 days after the subsequent resection has been made. The thick glass rod supporting the posterior wall holds it up and accomplishes diversion in a manner similar to the method described by Wangensteen,[12] who used two separate glass rods.

Closure of this type of transverse colostomy has been, in our experience, much easier than is the case when the posterior wall of the bowel is divided.

We wish to report briefly our experience with 41 cases of acute complete obstruction of the left colon, drawn from our own practice and from the Surgical Service of the Massachusetts Memorial Hospital. The cause of the obstructions was primary carcinoma of the colon in 39 cases, recurrent advanced carcinoma

TABLE I

	No. of Cases
Left transverse colon	1
Splenic flexure	1
Descending colon	6
Sigmoid colon	27
Recto-sigmoid	6
Total	41

TABLE II

Complications of 24 cecostomies

	No. of Cases	Per Cent
Unsatisfactory decompression*	6	
Gross wound infection†	1	
Pelvic abscess	1	
Incisional hernia	1	
Total	9	37

* Requiring subsequent transverse colostomy in 3 and ileostomy in 1 case.
† Minor infections not included.

TABLE III

Complications of 20 Transverse Colostomies

	No. of Cases	Per cent
Wound infection	1	5

of the cervix in 1 case, and an inflammatory mass resulting from previous operation in 1 case. The level of the obstruction is indicated in table I.

The decompression procedure used was cecostomy alone in 19 cases; cecostomy with concomitant transverse colostomy in 1 case; cecostomy with subsequent transverse colostomy in 3 cases; and cecostomy with subsequent ileostomy in 1 case. In these last 4 cases, the second procedure was necessary because of failure of the cecostomy to effect decompression.

Transverse colostomy alone was done in 16 cases and concomitantly with

cecostomy in 1 case (same case as mentioned above) and in 3 cases following failure of cecostomy, giving a total of 20. One sigmoid colostomy was done.

The complications of the 24 cases of cecostomy and of the 20 transverse colostomies are shown in tables II and III.

In this series of 41 cases there were five deaths, a mortality rate of 12.2 per cent. The single patient treated by sigmoid colostomy died on the second postoperative day of unrelieved obstruction. Of 24 patients treated by cecostomy, 2 or 8.4 per cent died. Of 20 patients treated by transverse colostomy, 2 or 10 per cent ended fatally. However, 1 of these died of coronary thrombosis on the tenth postoperative day after an uneventful convalescence, and the second died of cardiac failure within 24 hours. This patient was a man of 81 years of age who was in extremely poor condition and probably would not have survived any operative procedure. Nevertheless, the principles which we considered vital in the safe management of these cases were not adhered to in this instance in that a vertical incision was used; exploration was done; and the operation was prolonged—lasting 1 hour and 25 minutes.

SUMMARY AND CONCLUSIONS

Acute obstruction of the left colon is a serious surgical emergency requiring prompt surgical decompression.

The significance of the *closed loop* obstruction, which exists in most cases, is emphasized.

The advantages of a simple loop colostomy placed in the right half of the transverse colon, as the decompression procedure of choice, are discussed.

An analysis of 41 cases is offered in support of this opinion.

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INTESTINAL OBSTRUCTION AS A COMPLICATION OF URETEROSIGMOIDOSTOMY

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Uretero-intestinal anastomosis is beset with many complications peculiar to this type of surgery, as well as by the usual possible problems following any major surgical procedure. Of the former type of complications, those related to the kidney and ureter and to electrolytic changes have justly received the most attention as they are, numerically, the most important. There is, however, a third group of specific complications which has been relatively neglected in the literature, namely, those related to the intestines. In particular, the possibility of development of intestinal obstruction has received little attention. Since it is frequently life-saving to make an early diagnosis when intestinal obstruction is present, this paper is an attempt to emphasize this problem.

Jacobs and Stirling,[8] in reporting late results of ureterocolic anastomosis, found that 41.4 per cent of 1673 cases had complications. Of this group 17 patients, or 1 per cent of the total cases, developed acute intestinal obstruction. This complication occurred almost as often as renal calculus, fecal fistula and pyelonephritis alone without urinary obstruction. It is quite possible that some of the cases of fecal fistula were secondary to partial intestinal obstruction, as it was in one of our cases. Also significant in this report was that paralytic ileus or peritonitis with resultant abdominal distention occurred in 204 cases, or 12.2 per cent. Since these common complications could be mistakenly diagnosed when intestinal obstruction is present, this latter possibility should always be kept in mind.

We have recently encountered two types of intestinal obstruction following ureterosigmoidostomy which are rarely seen and which are reported in detail to stress this problem. These are cases of volvulus and intussusception.

CASE REPORTS

Case 1. H. H., a 51 year old white man, was admitted on July 7, 1949, to the Atlanta Veterans Administration Hospital complaining of hematuria of six months' duration.

Diagnosis was infiltrative transitional cell carcinoma of the bladder located on the posterior wall just superior to the interureteric ridge. The past history revealed constipation for 10 years, which was relieved by various cathartics. A diagnosis of megacolon had been made elsewhere in 1944. Barium enema at this hospital in July 1949, had shown an area of constriction of the proximal end of the sigmoid but no dilatation of the bowel.

Since the findings in the sigmoid were not considered significant, on July 22, 1949, a bilateral ureterosigmoidostomy of the Cordonnier type[3] was done. Total cystoprostatectomy was planned for a later date. The postoperative course was completely uneventful for

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four days, the urinary output being 1200 cc., 2400 cc., 3200 cc., 2100 cc., and the nonprotein nitrogen being 41 mg. per cent on the third day as compared to 38 mg. per cent preoperatively. On the morning of the fourth day, while sitting at the side of the bed for the first time, the patient experienced a sudden desire to defecate and micturate and felt some cramping lower abdominal pain. When he was returned to bed the pain markedly intensified, being unrelieved by narcotics. Pulse, respiration, and temperature all immediately increased. The abdomen was only very slightly distended, but peristalsis was absent whereas it had been active on the previous day.

Roentgenograms of the abdomen showed 1 to 2 inches of free air under the diaphragm and large dilated loops of colon with only minimal air in the small bowel loops. A mistaken diagnosis of paralytic ileus was made, the free air being thought to be due to the operative procedure. Conservative therapy with Harris tube suction, blood transfusions, oxygen tent, digitalization and supportive measures was followed.

In 24 hours the condition of the patient worsened. He now demonstrated an abdominal fluid wave. Roentgenograms showed the same amount of free air with more distention of the large bowel but none of the small bowel. In view of these facts, a diagnosis of volvulus of the sigmoid was considered probable and the patient was reoperated upon under sodium pentothal and local block novocaine anesthesia.

At operation the peritoneum contained approximately 2000 cc. of urine and generalized peritonitis was present. A volvulus of the sigmoid was found, occurring between the two sites of ureteral anastomosis. The left ureterosigmoidostomy had completely disrupted, and the ureter was lying free in the peritoneal cavity. Severe large bowel distention had apparently caused the separation of the anastomosis. The right anastomosis was intact and was functioning well. The volvulus was reduced; the opening in the sigmoid was closed; the gas in the large bowel was evacuated through the rectal tube; and a left cutaneous ureterostomy was made.

Postoperatively, the temperature was 105F. rectally and the patient presented a picture of profound toxemia. The temperature gradually lowered, but the urinary output remained poor, averaging 600 cc. daily from the left cutaneous ureterostomy and 25 to 100 cc. from the rectal tube. It was believed that the right ureter was probably at least partially blocked. The nonprotein nitrogen rose to 256 mg. per cent by the fifth day following the second operation. This was thought to be caused by severe pyelonephritis and toxic nephritis. On the fourth and fifth days, hemorrhage occurred from all the tubes, suggesting the hepatorenal syndrome. The patient became more comatose and died on the fifth day.

Autopsy showed acute passive congestion of the esophagus, heart, lungs, liver, kidney, brain and bladder, adrenal cortical atrophy and acute bilateral pyelonephritis with sclerosing papillitis as well as the previously known carcinoma of the bladder.

Case 2. R. S. W., an 11½ month old white male, was admitted on August 12, 1951, to the Henrietta Eggleston Hospital with a diagnosis of exstrophy of the bladder.

He had a bilateral uretero-intestinal anastomosis of the Cordonnier type[3] done on Aug. 28, 1951. The postoperative course was uneventful for the first nine days, after which he began to vomit all ingested foods. His abdomen rapidly distended and attempts to relieve this distention by rectal tubes, nothing by mouth, prostigmine, and other usual methods were unsuccessful. The vomiting continued; the abdomen became more distended; and there was hypoactive peristalsis without rushes.

A flat roentgenogram of the abdomen was taken, and from this it was thought that a small bowel obstruction was present. A Miller-Abbott tube was passed into the stomach. At the end of 72 hours, the patient's abdomen was still partially distended but the tube ceased to advance. During this period, the patient had had only intravenous fluids, penicillin, streptomycin and one blood transfusion. His blood chlorides and serum proteins were within normal limits. A diagnosis of small bowel obstruction probably due to adhesions was made.

Seven days after the onset of signs and symptoms of obstruction an exploratory laparotomy was done. A jejunal intussusception was located approximately 5 feet below the duode-

num. The Miller-Abbott tube had advanced to this point. One small perforation in the involved segment was found and this was closed in the usual manner. The intussusception was reduced. The bowel appeared to be viable at all points. The tube was allowed to remain in place for 72 hours, after which the patient was started on surgical liquids. Following this, he made an uneventful recovery and was dismissed on October 2, on the twelfth postoperative day.

DISCUSSION

In addition to the usual complications occasionally encountered following any major surgical procedure the performance of ureterosigmoidostomy involves further dangers which might be separated artificially into biochemic, urologic and intestinal categories. All three of these special groups of complications, however, are closely interrelated.

The biochemic and urologic types are more common and usually occur as delayed complications although they can develop in the immediate postoperative period. Primary electrolytic or biochemic changes which appear after diversion of the urine to the colon are hyperchloremic acidosis[6, 2] and, less commonly, hypopotassemia.[5] Urologic complications include ureteral reflux,[1] ureteral meatal stenosis,[7] ureteritis,[4] ureterectasis,[4] hydronephrosis,[4, 8] calculi,[8] cortical abscesses[4] and pyelonephritis.[8] Urinary leakage at the site of anastomosis also may result in a urinary fistula.[4] All of these lead to possible renal insufficiency and uremia with secondary electrolytic changes as contrasted to the primary electrolytic changes resulting directly from diversion of the urinary stream to the bowel.

The primarily intestinal complications are less common but are significant because surgical intervention is frequently indicated as a life-saving measure. These complications are very apt to occur in the immediate postoperative period but may develop later. Included in this grouping are paralytic ileus,[8] chronic diarrhea with possibility of development of hypopotassemia[5] and rectal incontinence, intestinal obstruction[8] and fecal leakage at the site of ureterosigmoid anastomosis[8] resulting in localized abscesses, fecal fistulas, or generalized peritonitis. It is probable that some cases of fecal fistula are secondary to intestinal obstruction rather than to leakage of the anastomosis.

The most frequent causes of intestinal obstruction following ureterosigmoidostomy are fibrous bands and herniation of a loop of intestine behind the necessarily fixed sigmoid colon. Our case of fecal fistula was secondary to herniation of intestine with partial intestinal obstruction. Attempt has been made to lessen this possibility by suturing the sigmoid colon between the anastomoses to the posterior peritoneum but herniation can still occur inferior to the anastomosis. Chronic partial intestinal obstruction frequently can result from fibrous bands and adhesions.

The two complications described here in detail admittedly are rare. Jacobs and Stirling[8] observed 1 case of volvulus in the 1673 cases. As far as can be determined, this is the first case of intussusception reported as a complication of ureterosigmoidostomy. The volvulus occurred as a direct result of the necessary fixation of the sigmoid. The intussusception was a more distant complication, apparently not related to the anastomosis itself, but it probably developed be-

cause of the necessary manipulation of the intestine. In as much as ureterosigmoidostomy frequently is done for exstrophy of the bladder during the period when a child is more prone to develop intussusception, this possible complication should be borne in mind.

SUMMARY

Two unusual cases of intestinal obstruction following ureterointestinal anastomosis are related in detail.

These cases are reported primarily to emphasize the necessity for recognition of intestinal obstruction as a distinct complication of this procedure. Abdominal distention, cramping and ileus which occur commonly after ureterosigmoidostomy should be watched carefully for signs of this more serious complication.

Other complications, grouped artificially into intestinal, urologic and biochemic categories, are mentioned briefly for completeness. These have been extensively discussed in other papers.

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THE ACQUIRED SHORT FOURTH METATARSAL

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INTRODUCTION

Interest in the short fourth metatarsal was first brought to the attention of one of us (W. L. M.) in 1943 by Doctor Robert A. Knight of the Campbell Clinic, Memphis, Tennessee. Clinical curiosity stimulated us to seek the reason for the peculiar predilection to the fourth metatarsals, especially in children with paralytic poliomyelitis and spastic paralysis. In the flail foot and ankle, all metatarsals show a delay in growth with a resultant short foot. In some feet, the third, fourth and fifth metatarsals will be retarded in growth, but frequently, the fourth alone will be affected. The authors observed an early closure of the epiphyseal plate, but the exact reason for this premature closure has not been discovered.

It is believed that the introduction or discussion of this oddity will stimulate interest and more widespread study and observation. The authors were unable to find literature describing the acquired short fourth metatarsal in poliomyelitis or spastic paralysis, although Hooker,[4] Stern[6], and Stiles[7] have described the congenital short fourth metatarsal.

A review of the various textbooks on the time of closure of the metatarsal epiphyses gave no information on the order of closure, only the fact that under normal conditions, the epiphyses close from around the sixteenth to the eighteenth year; a year or two earlier in females.

A total of 2,682 examinations have been made in the outpatient clinic of the Carrie Tingley Hospital for Crippled Children since July 1949. Among these, 33 patients with short fourth metatarsals were observed. Of the 262 poliomyelitis patients with paralysis of the lower extremities, 16 had short fourth metatarsals. Of 192 cerebral palsy patients of all types with hemiplegia or quadriplegia, 9 of the spastic type had short fourth metatarsals. Of 121 patients with congenital anomalies of the lower extremities, five short fourth metatarsals were found. Of 20 patients with burns of the lower extremities, 18 with osteomyelitis of the lower extremities, and 35 with rheumatoid arthritis, three short fourth metatarsals were found, one in each of the three categories. Of the 16 poliomyelitis patients who had short fourth metatarsals, the onset of the disease was 5 months of age for the earliest and 7 years for the latest, with the average age at the onset at 2.1 years. One patient had osteomyelitis of the fourth metatarsal at the age of 3 years. Destruction of the epiphysis in this case explains the shortness of the fourth metatarsal (table I).

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TABLE I

Short fourth metatarsal study

Poliomyelitis Patients

Patient	Age	Onset	Extremity Involved (Lower)	4th Metatarsal Right (in cm.)	Length Left	Difference Between Short & Normal 4th (in cm.)
I. L.	17	1 yr.	Bilateral	4.8	4.5	
A. K.	15	14 mo.	Left	6.9	4.4	2.5
M. G.	12	5 yr. 5 mo.	Right	4.6	5.4	0.8
J. M.	10	2 yr.	Right	4.0	5.1	1.1
D. B.	6	6 mo.	Bilateral	3.2	3.4	
C. G.	13	1 yr. 6 mo.	Bilateral	3.9	5.9	2.0
P. C.	12	2 yr.	Bilateral		4.4	
G. M.	16	3 yr.	Left	6.0	4.6	1.4
E. L. C.	15	2 yr. 6 mo.	Bilateral	5.2	4.3	
E. J.	15	3 yr.	Bilateral	4.1	5.0	0.9
W. E. B.	15	2 yr.	Bilateral	Films Not Available		
B. B.	7	5 mo.	Right	3.9	4.3	0.4
O. G.*	15	3 yr.	Bilateral	4.7	4.7	
C. G.	19	8 mo.	Bilateral	4.5	4.6	
J. L. B.	16	2 yr.	Left		4.6	
N. B.	16	3 yr.	Left	6.2	4.5	1.7

Cerebral Palsy Patients

Patient	Age	Diagnosis	Extremity Involved	4th Metatarsal Right (in cm.)	Length Left	Difference Between Short & Normal 4th
E. B.	17	Paraplegia	Bilateral	4.6	4.6	
L. Y.	5	Hemiplegia	Left	4.0	3.9	0.1
M. L. E.	13	Quadriplegia	Bilateral	4.0	4.7	0.7
P. P.	19	Quadriplegia	Bilateral	6.3	6.1	0.2
C. L.	5	Hemiplegia	Right	Films Not Available		
G. C.	13	Hemiplegia	Left	5.7	5.5	0.2
D. B.	11	Quadriplegia	Bilateral	4.3	4.3	
J. S.	12	Quadriplegia	Bilateral	Films Not Available		
A. L. M.	14	Hemiplegia	Right	5.1	6.7	1.6

Miscellaneous

Patient	Age	Onset	Diagnosis	4th Metatarsal Right (in cm.)	Length Left	Difference Between Short & Normal 4th
A. B.	13	13 yr.	Rh. Arth, Left	8.0	6.1	1.9
E. P.	10	1 yr.	Burn Con., Left	5.8	5.0	0.8
V. O.	19	Birth	Left Hip Disl.	6.0	3.5	2.5
V. L. R.	9	Birth	Clubfoot, Right	4.5	4.7	0.2
F. D.	17	Birth	Clubfoot, Right	5.9	7.1	1.2
F. C.	5	3 yr.	Osteo., Left		5.0	
C. E.	17	Birth	Cong. Anom., Right	Films Not Available		
A. B.	12	Birth	Cong. Anom., Bilat.	Films Not Available		

* See figure 1.

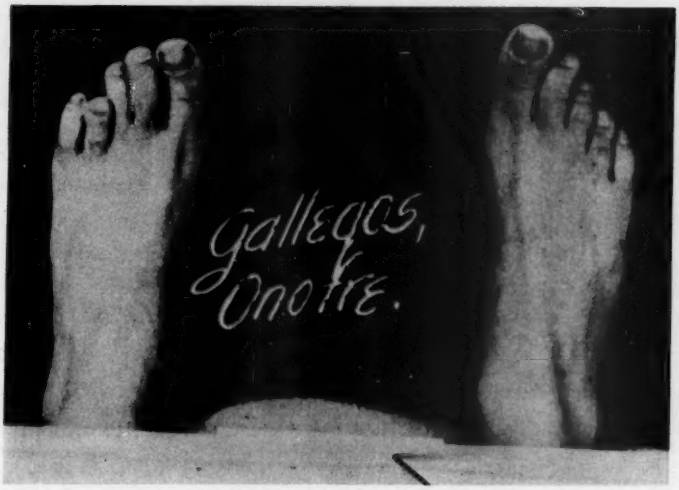

FIG. 1. O. G., male, aged 15. Poliomyelitis onset was at the age of 3 years. Note the relative shortening of the fourth and fifth metatarsals on the right and the fourth metatarsal on the left.

ETIOLOGY

Many questions remain unanswered: Why is the fourth metatarsal frequently the one with congenital shortening? Is there some genetic peculiarity that makes the fourth metatarsal susceptible to acquired shortening in patients with poliomyelitis and cerebral palsy?

In poliomyelitis, the epiphyseal plates of the third and fifth metatarsals may also close prematurely, producing shortness of these rays. However, in patients having poliomyelitis and spasticity, if a single metatarsal is found shortened, it is almost invariably the fourth.

The mechanical theory is based on the fact that many of the children have borne weight in the equinovarus position and that, possibly, unusual stresses were placed on the epiphyseal plate of the fourth metatarsal, causing shortening. It was thought that the fourth metatarsal epiphyses were traumatized by faulty weight bearing, as well as the third and fifth metatarsal epiphyses in some cases. However, in our series, 2 of the children had valgus feet and, to our knowledge, had not borne weight in either the sitting or the standing position. This leads us to believe that there are other unknown factors involved. Vascular supply, nerve supply, endocrine influences, nutritional and metabolic factors were considered, but no peculiarities could be found that might account for premature closure of the plate.

SUMMARY

Thirty-three patients with acquired short fourth metatarsals have been observed.

The acquired short fourth metatarsal occurs most frequently in children with paralytic poliomyelitis and spactic paralysis. It also occurs in patients having osteomyelitis, severe burns, rheumatoid arthritis and epiphyseal trauma.

The etiology of the acquired short fourth metatarsal has not been proved conclusively. In poliomyelitis, the epiphyseal plates of the third and fifth metatarsals also may close prematurely, producing shortness. However, in patients having poliomyelitis and spasticity, if a single metatarsal is found shortened, it is almost invariably the fourth.

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ISLET CELL ADENOMA OF THE PANCREAS

A CASE REPORT

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A functioning islet cell adenoma of the pancreas is a rare tumor. The first report of a case with hyperinsulinism was made by Wilder and his associates in 1927.[12] Since that time, several reports have appeared in the literature of successful removal of these tumors.[5, 7, 11]

A survey of the literature revealed an incidence of this type of tumor of less than 1 per cent in over 27,000 cases studied at necropsy and 224 removed at operation.[6, 7, 9, 10, 13] Since the greater number of the Islets of Langerhans occur in the body and tail of the pancreas, the greater number of islet cell adenomas are found in these areas; about 25 per cent may occur in the head. In only 5 per cent of this group are the tumors multiple. Clinical hypoglycemia as a result of neoplastic proliferation pathologically may manifest itself in one of four ways: as benign adenomas, 70 per cent; as islet cell adenomatosis, 2 per cent; as metastasizing islet cell carcinomas, 8 per cent; and as a malignant but nonmetastasizing islet cell tumor, 20 per cent.[7]

The adenomas usually are well encapsulated, measuring about 1 to 2 cm. in diameter. The malignant tumors are on the average twice the size of the adenomas; are more vascular and more invasive. The insulin content of these tumors varies from 4 to 40 times that of the normal pancreas per gram of weight.[1]

Symptoms. The onset is usually insidious, appearing gradually over a period of weeks or months. Due to the hypoglycemia as a result of hyperinsulinism, the manifestations of the low blood sugar levels primarily are those affecting the sympathetic and central nervous systems. As a result of this diffuse stimulation, the symptomatology may be varied and bizarre in character; many patients being inaccurately diagnosed as being neurotic, emotionally unbalanced, or hysterical.

Those symptoms resulting from sympathetic nervous system stimulation are: pallor, flushing, nausea, hunger, epigastric pain, vertigo, cardiac palpitation, and syncope. Symptoms related to central nervous system disturbance usually occur from severe attacks and appear as slurring speech, diplopia, restlessness, tonic or clonic muscle spasms, convulsions, and in extreme cases, coma and death. The psychiatric manifestations, too, may be widespread, and these include disorientation, retrograde amnesia, negativism, mania, and apprehension.

Diagnosis. The chief diagnostic criterion is *Whipple's triad*, as follows: (1) at-

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tacks of insulin shock occurring during fasting or following severe mental or physical exercise, (2) blood sugar levels below 50 mg. per cent during attacks, and (3) prompt recovery following the administration of glucose. Exploratory operation is advised if a patient fulfills these criteria, providing careful studies have been made to exclude extrapancreatic factors, such as liver diseases with alteration in glycogen storage and disorders associated with hypofunctioning of the anterior lobe of the pituitary, adrenal cortex, and thyroid gland.

The glucose tolerance test may be an aid. Generally, in cases of hyperinsulinism, the curve includes a subnormal fasting blood sugar level with the peak rarely exceeding 120 mg. per cent, and a return of subnormal levels within two hours, with these levels persisting throughout the third to the sixth hours.

Treatment. Operation is definitely indicated for these patients fulfilling the *Whipple's triad.* Medical therapy continued over a long period of time may result in mental deterioration due to prolonged hypoglycemia; malignant changes may occur in a benign islet cell adenoma and obesity may develop as a result of the high carbohydrate diet to control the hypoglycemia state. Obesity makes surgical exploration much more difficult.

Careful search of the entire pancreas and adjoining areas for an adenoma can be made through a supra-umbilical transverse type of abdominal incision. Failure to discover a tumor in the body or tail, requires careful palpation and partial mobilization of the head of the pancreas for a thorough search of this area. Simple enucleation or excision of the tumor is the operation of choice. In the event no tumor is palpable, a partial pancreatectomy should be done. The value of this procedure has been shown by the results obtained by several surgeons.[2, 3, 4, 8]

REPORT OF CASE

Miss E. E. T., a 38 year old white woman, was admitted to this hospital on June 4, 1951. Her illness began in 1947 with the onset of episodes of weakness and sweating always occurring between 10:30 a.m. and 2:00 p.m. These symptoms were always relieved by drinking a glass of milk or eating a banana. The attacks increased in frequency and severity, and she experienced additional symptoms of light-headedness, numbness, nervousness, trembling, tingling of the extremities, severe hunger, inability to concentrate, and temporary loss of memory. Her blood sugar was 57 mg. per 100 ml. of blood during an episode on May 16, 1949. A physician diagnosed her condition as a functional hypoglycemia on an emotional basis. Her past history and inventory of the systems were essentially negative and noncontributory except for a gain in weight from 143 to 150 pounds.

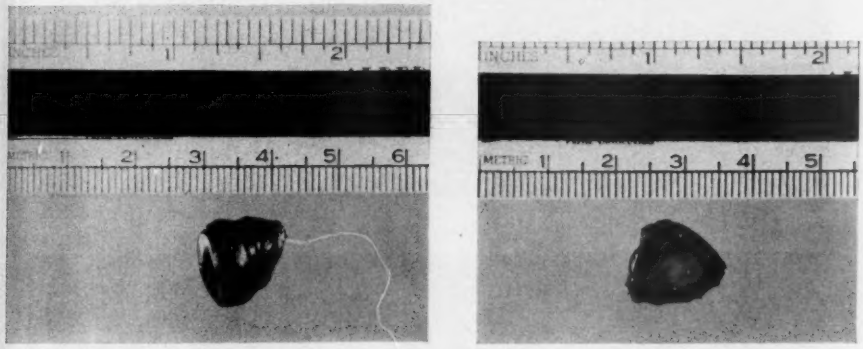
The physical examination revealed a well developed and well nourished white woman appearing to be in good health. Examination of the heart and lungs was negative. The neurologic findings were within normal limits.

The laboratory studies, including a complete blood count, urinalysis, and examination of hepatic, renal and adrenal functions were all within normal limits. The basal metabolic rate was 15 per cent below the normal standard. The fasting blood glucose was 60 mg. per 100 ml. of blood. The intravenous six hour glucose tolerance test showed a typical hypoglycemic curve as follows: fasting blood glucose was 101 mg. per 100 ml. of blood, one-half hour blood specimen was 121, one hour specimen was 104, two hour specimen was 65, three hour specimen was 42, four hour specimen was 40, five hour specimen was 28, and the six hour specimen was 53 mg. of glucose per 100 ml. of blood. A similar response was obtained following an oral six hour glucose tolerance test. Prolonged fasting for 19 hours resulted in a blood glucose level of 26 mg. per 100 ml. of blood. This was accompanied by typical hypoglycemic

symptoms which immediately were relieved by the intravenous injection of 50 ml. of 50 per cent glucose.

A diagnosis of islet cell adenoma of the pancreas was made preoperatively, and on Aug. 17, 1951, an exploratory laparotomy was done using a left transverse supra-umbilical incision. There were filmy adhesions in the upper abdomen between the liver, gallbladder and duodenum, and between the posterior wall of the stomach, transverse mesocolon, and the head of the pancreas. There was a circumscribed, well encapsulated, slightly movable, red-brown mass, measuring 1 cm. in diameter deep in the inferior portion of the head of the pancreas. It was anterior and to the right of the superior mesenteric vein and just above the third part of the duodenum. This adenoma was shelled out without difficulty.

Pathologic Examination. The specimen consisted of purplish-red tissue measuring 1 cm. in diameter and 2 cm. in length, with a smooth and faintly lobulated surface. The cut surface revealed pink tissue in which there were nodular areas



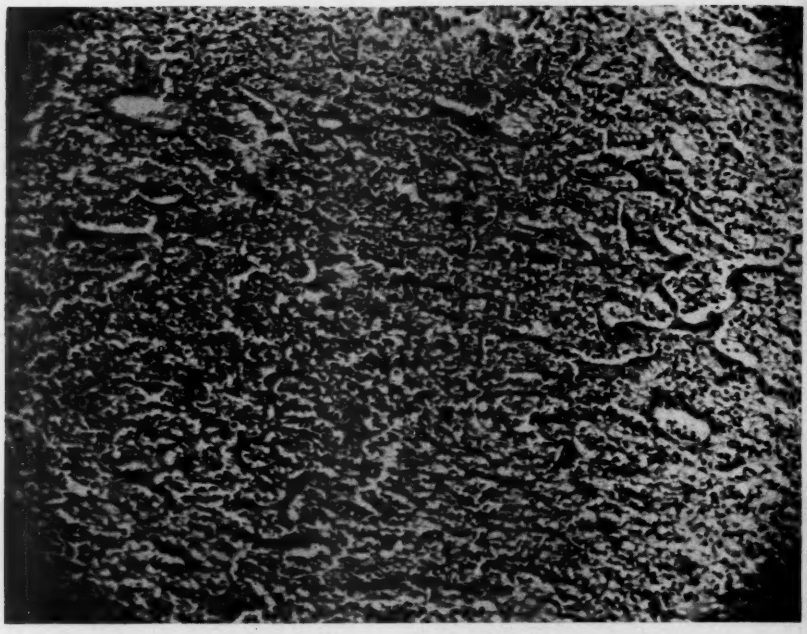
a b

FIGS. 1a and b. (a) Circumscribed and encapsulated tumor, smooth and faintly lobulated. In (b) on cut section the central portion appeared as a nodular gray and translucent area and the periphery of pink tissue.

of gray translucent tissue (figs. 1a and b). Microscopic examination showed a very cellular tumor composed of polyhedral cells arranged in cords and pseudo-rosettes in a delicate reticular stroma. The cells contained eosinophilic granular cytoplasm and a centrally placed round or oval nucleus; the basophilic nuclei were densely granular; small acini were formed occasionally; the stroma was very vascular; the tumor was encapsulated; no malignancy was seen (figs. 2a and b).

Pathologic Diagnosis. Islet cell adenoma, benign, head of pancreas.

The patient was given 1000 ml. of 5 per cent dextrose in one half strength physiologic sodium chloride solution during the operation. The blood glucose was 155 mg. per 100 ml. of blood during the operation. The postoperative course was uneventful. The fasting blood glucose ranged between 84 and 121 mg. of glucose per 100 ml. of blood. An hourly blood glucose curve during a 24 hour fast on Sept. 25, 1951, was flat with no readings below 89 mg. of glucose per 100 ml. of blood. She no longer experienced episodes of hypoglycemia. She returned in December 1951 and November 1952 and was found to be in excellent health with no recurrence of symptoms.



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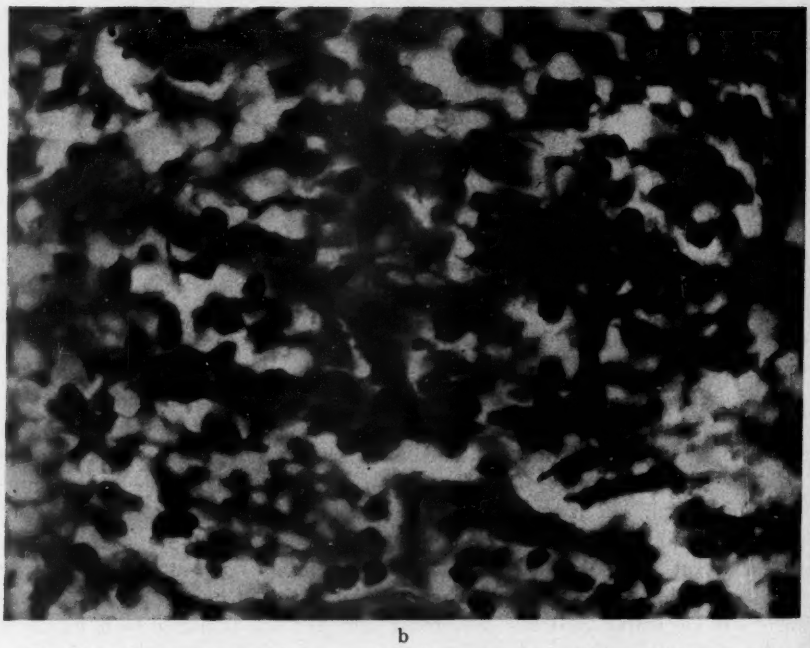

b

FIG. 2 (a). Low power revealed a very cellular tumor with cells arranged in cords and pseudorosettes in a delicate reticular stroma.
(b) High power revealed polyhedral cells with densely stained round or oval nuclei.

SUMMARY

The incidence, pathology, symptomatology, and treatment of islet cell tumors of the pancreas with hyperinsulinism are presented. Patients with symptoms resulting from hypoglycemia fulfilling the *triad* as outlined by Whipple, providing extrapancreatic factors have been ruled out, should be operated upon. Enucleation or excision of the tumor, if found, is the procedure of choice. In the event a tumor is not palpable in the pancreas, a partial resection of the pancreas is recommended. A report of a typical case with a benign islet cell adenoma of the head of the pancreas with successful surgical removal and immediate cure is presented.

Grateful acknowledgement is made to Dr. T. E. Corcoran, Chief, Department of Pathology, Veterans Hospital, for selection of the slides and pathologic report of the tissue in this case. The members of the Department of Medicine are complimented for their thorough and careful study of this patient.

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CHRONIC INTUSSUSCEPTING CARCINOID OF THE ILEOCECAL VALVE AND CECUM

A CASE REPORT*

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Lubarsch,[7] in 1888, first recognized a primary tumor of the gastrointestinal tract which occurred most commonly in the appendix and which microscopically resembled carcinoma but was distinctive in appearance and behavior. The name *carcinoid* was applied to this tumor in 1907 by Obernhorfer, who believed it to be benign. More recent studies[2, 4, 9] have emphasized that carcinoids are not necessarily of a benign nature. A recent survey by Pearson and Fitzgerald[12] revealed complete lack of regional node distant metastasis in carcinoids primary in the appendix (98 of 140 cases). However, a few cases of carcinoid of the appendix with metastasis have been reported.[1, 16] In the nonappendiceal group (30 of 140 cases), metastases were present in 38 per cent. The most common sites of distant metastases include the mesenteric nodes, liver, omentum, distant nodes and bones.

The incidence of all carcinoids discovered at autopsy varies from 0.34 per cent[14] to 1.8 per cent[15] with a predominance of appendiceal lesions. In the nonappendiceal group, the ileum is the commonest site. Carcinoid has been found in the stomach, gallbladder, cecum, colon, ampulla of Vater, Meckel's diverticulum, rectum and teratoma of the ovary.[10, 13, 3, 5] One case of carcinoid of the ileocecal valve with intermittent obstruction is reported by Pearson and Fitzgerald.[12] No mention of intussusception was made in this case. Carcinoid may occur at any age; appendiceal carcinoids are more frequently discovered in the third decade and the nonappendiceal lesions in the middle age group.

The signs and symptoms, when present, are those of intestinal obstruction when the tumor is in the bowel, and of inflammation when it is in the appendix. The incidence of symptoms in the nonappendiceal group varies with each series,[4, 6] with a probable average of about 20 per cent.

Without the symptoms suggestive of obstruction, the diagnosis of small bowel lesions is difficult. Occasionally the diagnosis may be made roentgenologically by the appearance of kinking of the small bowel in the absence of an intrinsic tumor.[9] The diagnosis in appendiceal lesions is practically impossible since the clinical findings are essentially those of appendicitis.

The case to be presented is an unusual carcinoid.

CASE REPORT

This 57 year old white woman was admitted to The Spartanburg General Hospital on Nov. 22, 1952 because of a mass in the right abdomen which was discovered by her physician

* From the Department of Surgery of The Spartanburg General Hospital and The Wallace Foundation.

a short time prior to admission. No symptoms were present which were attributed to the mass. The patient had not lost weight.

Past History: This patient had a hysterectomy and appendectomy in 1938. In 1951 a diagnosis of gallstones was made. There was also a history of three rectal operations for stricture. The family history was essentially negative.

Physical Examination: The patient had the appearance of a middle-aged white woman, somewhat pale, lying comfortably in bed. Examination of the head, neck, eyes, ears, nose and throat revealed nothing of importance. The heart and lungs were grossly normal. The blood pressure was 135/76 and the pulse regular at 80 per minute. Examination of the abdomen revealed moderate obesity; and the presence of a lower midline scar was noted. A slightly tender mass, 8 by 10 cm. in size, was noted in the upper portion of the right lower quadrant, approximately midway between the umbilicus and anterior iliac spine. There was moderate fixation of the mass. Peristalsis was normal. No other masses were noted. The

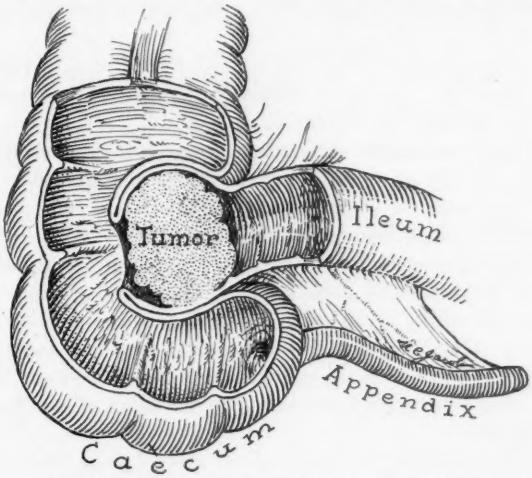

FIG. 1. Chronic intussuscepting carcinoid of the ileocecal valve and cecum

cervix was of normal consistency and color. No adnexal masses were noted. There was considerable scarring at the anal orifice and moderate sphincter spasm.

Initial laboratory studies were essentially normal. A barium enema obtained on November 24 showed "no intrinsic lesion of the colon. There is a defect intermittently evident in the medial aspect of the cecum due to pressure from a neighboring abdominal mass."

After adequate preparation, exploration through a long right rectus incision was made. A tumor involving the cecum and ileocecal valve was found. Two very small elevated nodules were palpated in the dome of the liver. The right colon, including the lower 12 inches of the ileum and corresponding mesentery, was resected, followed by an end to side ileotransverse colostomy. It was not deemed advisable to remove the nodules in the liver due to relative inaccessibility.

Following operation, gross examination of the specimen revealed the presence of an intussusception of the ileum of approximately 5 cm. in length into the cecum. Upon opening the intussusceptum, a tumor was noted. After incising the lower ileum, the tumor was found at the ileocecal valve and submucosal in position. Extrinsically necrotic fat tissue bound by adhesions to the ileocecal valve was also noted. A pathologic diagnosis of "argentaffinoma of the ileocecal valve, intussusception of the cecum and gangrene of fat tissue" was reported (fig. 1).

The postoperative course was uneventful. A Harris tube was removed on the fourth postoperative day and oral feedings were begun. The highest temperature reading was 99.4F. She was discharged in good condition on the seventeenth postoperative day. Subsequently her condition has been good.

DISCUSSION

In the case presented, it was difficult to see how a lesion of this type failed to produce symptoms of intestinal obstruction in a region normally narrowed by the ileocecal valve. Complete invagination of the ileum into the cecum and ascending colon was evidently prevented by the inflammatory reaction about the gangrenous fat tissue which acted as a natural barrier to continued intussusception. Preoperatively, the barium enema was more suggestive of an extralumenal mass such as lymphosarcoma. An intussusception was not apparent in the roentgenogram.

An opportunity to inspect the small nodules in the liver in this case will be afforded at a later date, since a cholecystectomy is contemplated. If possible, these nodules will be removed for microscopic study.

Intussusception from benign tumors of the terminal ileum is quite common. Olson, Dockerty and Gray[11] found a high incidence of intussusception (78.6 per cent) in a group of patients with obstruction. The report of a case of carcinoid in the ileocecal valve with intussusception has not been noted in the literature.

There is general agreement that carcinoids are slow-growing, both at the site of origin and in the metastatic lesions. The classical case of Mallory[8] which had *inoperable* carcinoid of the ileum with retroperitoneal metastasis is one example of this tendency. After 20 years, the metastatic lesion was found to be essentially unchanged at autopsy. Some patients with carcinoids have been observed, however, who died only a few years following the onset of symptoms with generalized metastases.[12]

Because of the malignant tendency of carcinoid lesions, the choice of treatment is, in our opinions, wide resection of the involved tissue and isolated metastases, if feasible. Roentgenotherapy is probably of little or no value.

It is apparent from experience with lesions in the ileocecal region that it is not always possible to determine preoperatively or during exploration the exact nature of the disease. With a conservative approach to lesions of doubtful etiology in this area, the presence of a true malignancy may occasionally be missed.

SUMMARY

An unusual case of carcinoid involving the ileocecal valve with intussusception is reported.

Some clinical features of this disease are presented.

Radical excision is advised for carcinoid tumors of the ileocecal region.

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VESICAL CALCULI FOLLOWING SUPRAPUBIC PROSTATECTOMY

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In 1936 Lett[4] listed 12 instances of vesical stones following prostatectomy. Prior to this time Caravan and Lourdel as quoted by Blum and Rubritzius[1] reported two occurrences in 213 operations. Since Lett's paper only sporadic cases with postprostatectomy calculi are mentioned in the literature.[2, 3, 5] The most important cause for stone formation was considered to be bladder neck or urethral obstruction with a resultant urinary stasis and calculous deposit on a piece

TABLE 1

Causes of vesical calculi following prostatectomy

1. Bladder neck obstruction.
2. Urethral strictures and membranes; meatal stricture.
3. Residual and recurrent prostatic adenomas or carcinoma.
4. Foreign bodies introduced at the time of operation:
   a. gauze
   b. rubber tubing
   c. instruments, needles
   d. oxycel
   e. sutures
5. Renal or ureteral stones passed after operation into the bladder.
6. Retained prostatic chips following a transurethral resection.
7. Calculi formation in a diverticulum.
8. Dislodged prostatic stones or fragmented vesical calculi at the time of operation.
9. Infection with formation of inflammatory debris and slough.
10. Neurogenic and atonic myogenic bladders.

of inflammatory debris. Other etiologic factors are listed in table 1. There have been cases reported, however, in which no obstruction was present and no definite etiology could be discovered.[6]

At the Beth Israel Hospital 9 of 360 patients who had suprapubic transvesical prostatectomy in a two year period (1950 and 1951) developed bladder stones 4 to 18 months postoperatively. Thus the incidence at this hospital of postprostatectomy (suprapubic) calculi was 2.5 per cent. This was a large increase in the occurrence of this type of calculi since only 2 cases were found in the preceding four years. The 9 patients had suprapubic cystolithotomies. A study of these patients was undertaken to determine possible causes of the rapid and relatively frequent incidence of stone formation. The surgery in the 360 patients was done by eight members of the urological staff—all using similar technic for most of the operations.

In the same two year period two other suprapubic cystolithotomies were done on 2 patients who had had suprapubic prostatectomies a few months be-

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fore at different hospitals. These 2 patients had developed obstructions in the posterior urethra (table 2).

TABLE 2

Cases with obstruction

Patient & Age	Interval Following Prostatectomy	Hemostatic Agent	Type of Obstruction	Nidus of Stone	Urine Culture
S. R. 67	1 year	Ribdam repacked with gauze on operation day	Stricture of entire posterior urethra	None	B. pyocyaneous
S. V. 62	4 months	Gauze	Recurrent adenocarcinoma of prostate	None	Paracolon bacillus
M. S. 58	1 year	Ribdam	Residual prostatic calculi & adenomas	None	Paracolon bacillus
Suprapubic prostatectomy done at other hospitals					
A. L. 59	6 months	Pilcher bag	Stricture membranous urethra	None	Coliform organisms B. lactis aerogenes
P. R. 63	5 months	Gauze	Membrane across the mid-posterior urethra	Large protein center	

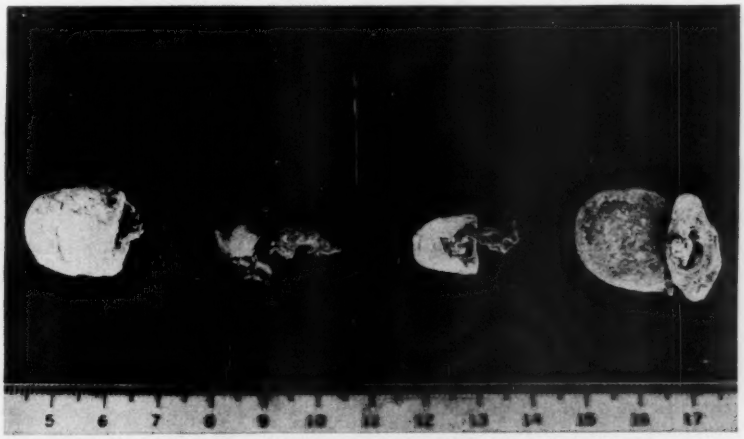

FIG. 1. Photograph of 4 stones from cases without obstruction demonstrating the protein centers.

Three of the 9 Beth Israel Hospital patients developed postoperatively, an obstruction in the posterior urethra causing residual urine and thus favoring the development of the calculi (table 2). The remaining 6 patients revealed no evi-

TABLE 3

Cases without obstruction—no residual urine

Patient & Age	Interval Following Prostatectomy	Hemostatic Agent	Urine Culture	Postop. Hospitalization	Stone Center	Other Factors
J. R. 74	4 months	Ribdam	Coliform bacilli	26 days	Strands of protein in 2 calculi	Few small prostatic calculi in surgical specimen
I. M. 74	12 months	Ribdam	B. lactis aerogenes	14 days	Large protein center in 2 calculi	
A. I. 76	6 months	Ribdam	Paracolon bacilli	15 days	Large protein center	4 calculi (ammonium urate calcium phosphate) at time of first stage prostatectomy
I. K. 61	6 months	Ribdam	Coliform bacilli nonhemolytic streptococci	18 days	Large protein center	
J. S. 62	4 months	Ribdam	Coliform bacilli B. lactis aerogenes B. Proteus	16 days	Large protein center	1 Jackstone calculus (calcium oxalate) removed at time of first stage prostatectomy
H. E. 78	18 months	Ribdam	Coliform bacilli	12 days	None	Calculus extended into prostatic fossa—10 calculi (calcium phosphate, ammonium magnesium phosphate) removed at time of first stage prostatectomy
	Average 8 months			Average 16.8 days		The three 2 stage prostatectomies had calculi removed at time of first stage

dence of bladder neck or urethral obstruction following the prostatectomy and the bladder contained no residual urine. The prostatectomies in these patients were all done through a suprapubic approach (three in two stages) with ribdam rubber packing packed in the prostatic fossa around a no. 22 F. Foley catheter. The adenomas removed were moderately large, averaging 54 Gm. The impressive finding in these 6 cases was that five of the six calculi contained centers of foreign material. The centers consisted of a large putty-like material; grey in color; from 1 to 3 cm. in diameter—except in one— in which only strands existed in the center (fig. 1).

Interest centered around the cases with the nidus centers since they composed a majority of the calculi in the patients without urethral obstruction. It was first demonstrated by gross and microscopic chemical tests (lithium chloride and iodine stain) that gauze strands did not compose any part of the foreign material. The material gave a positive test for protein by the Zanthoproteic and Millon methods. Microscopic sections demonstrated that the centers consisted of tissue containing a few nuclear-like remnants and were infiltrated with numerous bacteria. Stains for fibrin were negative.

I believe that this protein substance most likely represents slough from the prostatic bed. It is also possible that fibrin from old blood clots could have been the parent material since washing in urine changes the character of fibrin protein, resulting in a negative fibrin test. The most likely nidus is slough, because blood protein derivatives are more readily softened and fragmented by constant bathing in urine. Only one calculus in the 5 cases with bladder neck obstruction contained the same type of protein center.

The calculi in every case were the usual alkaline type associated with infection or foreign bodies. They were all composed of calcium phosphate and ammonium magnesium phosphate. Two calculi contained, in addition, small quantities of calcium carbonate and three contained in addition traces of ammonium urate. The patients had received postoperatively numerous courses of the available antibiotic and chemotherapeutic agents, but pyuria and positive urine cultures persisted. The urine cultures at the time of cystolithotomy revealed a variety of organisms (tables 2 and 3). Only 1 case in the nonobstructed group contained prostatic calculi. There were no diverticula of the bladder found in this series.

DISCUSSION

The postoperative histories of the nonobstructed patients were reviewed, but no unusual incidents or difficulties were encountered. In recent years the technic of hemostasis in most of the suprapubic prostatectomies was changed at this hospital from packing with gauze without using a urethral catheter, to packing with ribdam around a no. 22 F. Foley catheter. The suprapubic tube is often removed from the second to the fourth postoperative day and the Foley catheter used until the seventh to ninth postoperative day. The Foley catheter, which provides much less drainage than a straight catheter or suprapubic tube, may also prevent slough, old clots, or inflammatory debris from leaving the

bladder in which case the retained debris may form predisposing calculi. The high incidence of large protein nuclei (5 out of 6 in the nonobstructed cases) lend support to this view. Also, few calculi occurred when the suprapubic tube was relied upon for drainage during the first post-operative week as in previous years. As a measure to prevent future occurrences of calculi it is recommended that more thorough irrigation be used to remove clots, tabs, slough and debris before removing the catheter. At the time of operation, all adenomatous nodules should be carefully removed from the prostatic fossa and all tabs of tissue, especially at the bladder neck, should be trimmed away.

SUMMARY

Nine of 360 patients having suprapubic prostatectomies at the Beth Israel Hospital in the last two years developed, postoperatively, bladder calculi—an incidence of 2.5 per cent.

Three of the 9 patients developed postprostatectomy urethral obstructions which probably accounts for the stone formation. In the other 6 patients, however, there was no obstruction and no residual urine. Five of these 6 patients developed calculi with large protein centers. The protein centers are believed to represent slough, blood clot, or inflammatory debris which was retained in the bladder because of inadequate drainage. The use of thorough irrigation in order to remove these particles is suggested before the removal of the catheters. In addition, all tabs of prostatic tissue should be carefully trimmed away at the time of operation.

I wish to acknowledge the aid of Dr. S. F. Wilhelm in preparing this paper and also to thank the members of the Urological Staff whose patients were utilized in this series.

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EDITORIALS

THE IMPORTANCE OF TOTAL THYROIDECTOMY FOR PAPILLARY CARCINOMA OF THE THYROID GLAND

Papillary carcinoma is the dominant pattern in more than one-half of all malignant neoplasms of the thyroid. In contrast to the more anaplastic and ominous thyroid carcinomas, this major fraction of papillary neoplasms is characterized by a low growth potential, and by regional spread through cervical lymphatics rather than by humoral routes. Even after inadequate surgical treatment, these lesions tend to be dilatory, seldom metastasizing below the clavicles until 5 to 15 years after the primary attack on the disease. The histology frequently reflects these biologic peculiarities, mimicking the parent tissue so faithfully as to have produced misconceptions indicated by the terms *aberrant thyroid rest* and *benign metastasizing struma*.

This sort of clinical behavior has produced an attitude of surgical conservatism which is mainly based on the failure of surgeons to secure long term follow-up examinations of patients with this neoplasm. Five-year control without recurrence as high as 80 per cent is reported, but similar figures for 10 and 15 years are seldom quoted, and are usually not more than 60 per cent.

Contributing to surgical restraint in the management of this most curable of all thyroid cancers is the anatomic division of the thyroid into two convenient, laterally placed lobes, connected by a narrow isthmus. *Total lobectomy* has the sound of a good operation for cancer, but the procedure is entirely inadequate by standards applied to other sites of visceral cancer. Adequate surgery for cancer should include extirpation of the entire organ of origin, as well as the regional lymphatics, whether the latter show gross evidence of involvement or not. Assuming the presence of papillary carcinoma without clinical evidence of lateral cervical node deposits, the minimum procedure should be a total, *extracapsular* thyroidectomy, combined with a regional dissection in the area of the thyroid. This area of dissection should be bounded laterally by the internal jugular veins, above by the cricoid cartilage and below by the suprasternal notch.

Approximately one in every four times this procedure has resulted in the histologic demonstration of unsuspected lymph node metastases, usually adjacent to the lower pole of thyroid lobes, or in pretracheal nodes. More important, in 15 per cent of such instances involved nodes will be found *contralateral* to the thyroid lobe in which the primary lesion is located. In 2 patients, multiple foci of intraglandular neoplasm have been demonstrated.

For the patient with proved metastases to lateral cervical nodes, block dissection of the involved side, combined with total thyroidectomy is the only measure to secure the best chance of cure. In two such instances in the past several years, the primary lesion was found as an occult focus contralateral to the side of clinically apparent involvement of neck nodes.

A study of the lymphatic drainage of the thyroid gland, with the potential routes for contralateral metastases, should constitute sound evidence for the necessity of total thyroidectomy for papillary carcinoma.

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THE CONTROL OF HEMORRHAGE BY BLIND GASTRIC RESECTION

The management of uncontrollable hemorrhage is, with rare exception, the responsibility of the surgeon. It is he who is best equipped to attack the bleeding point at its source. Such a duty can be confidently accepted when the source of the hemorrhage is apparent. When the site of the bleeding is obscure, the surgeon's difficulties are multiplied, but his responsibility is unchanged.

These statements are no less true when dealing with hemorrhage into the upper gastrointestinal tract. It again is the duty of the surgeon to prevent these patients from bleeding to death. The fact that most upper gastrointestinal hemorrhage will subside without direct operative attack intensifies the problem without altering the responsibility. An attempt must be made to select for operation in the first 48 hours only those patients whose hemorrhage will not subside on conservative management. This judgment is doubly difficult when the source of the hemorrhage is unknown. Indeed, some surgeons advise against a direct attack on the bleeding vessel unless a preoperative diagnosis can be established.

Being unable to accept the thesis that it is better for a patient to bleed to death from an unknown source than from a known source, we have used the same indications for operation in all patients with upper gastrointestinal hemorrhage.

Briefly stated, these indications include evidence that the hemorrhage is massive and from the upper gastrointestinal tract. Hematemesis, when present, is reasonable proof that the bleeding point is proximal to the jejunum. The blood dyscrasias can be ruled out immediately by appropriate tests. Any indication of bleeding from esophageal varices should be investigated at once. When there is reasonable doubt on this question, it can be settled by the bromsulfalein test, roentgenographic examination, and, if necessary, esophagoscopy. If these requirements are satisfied and bleeding continues after 48 hours of conservative care, surgical extirpation of the bleeding point is mandatory. When the rate of hemorrhage is in excess of 1500 ml. in 24 hours, the period of medical management is shortened.

If this program is followed, one will inevitably operate upon some patients without a definite preoperative diagnosis. This situation is of no great consequence if the hemorrhagic lesion is readily demonstrable at the time of laparotomy. There will be some instances, however, in which no source of hemorrhage can be found after careful exploration. It is a firm desire to avoid this

situation which has prompted some surgeons to delay operation until a diagnosis can be established.

It has been our experience that small mucosal erosions or *flat ulcers* of the gastric mucosa are the usual source of hemorrhage when there is no other demonstrable lesion at operation. These defects are not palpable; are often not visible by gastrotomy; and sometimes may be found in the excised specimen only after a thorough search. Excision of these bleeding points can best be assured by doing a high partial gastric resection. This of necessity must be a so-called *blind resection.*

It is not without reason to question the wisdom of inflicting such a major surgical procedure as partial gastric resection on a very ill patient without assurance that the bleeding point will be included in the resected specimen. It is true, on the other hand, that to close the abdomen without any definitive procedure often results in repeated and fatal hemorrhage during the postoperative period. The question hinges then on the results one can expect from blind gastric resection.

Scattered reports in the literature are inconclusive. Those who have used blind gastric resection in the emergency treatment of active upper gastrointestinal hemorrhage do not always give complete data as to the lesion resected or the percentage of patients in whom bleeding was successfully interrupted. For this reason recently we have reported our own experience with blind gastric resection.

In a series of over 250 gastrectomies for gastric or duodenal ulcers, only 18 were done for the emergency treatment of upper gastrointestinal hemorrhage. At laparotomy no bleeding lesion was demonstrable in 6 of the 18 patients. A blind gastric resection was done on 5 of these patients. In each instance the responsible lesion was apparent in the specimen removed, and hemorrhage was controlled. The sixth patient did not have a resection. After a negative exploration, including gastrotomy, the operation was terminated. A fatal hemorrhage occurred 48 hours later, and autopsy revealed a tiny mucosal erosion which could have been removed handily with a blind resection of the stomach.

This small series of patients takes its significance from the fact that following the above program over a period of three years, 100 per cent of the patients operated upon for upper gastrointestinal hemorrhage were amenable to surgical control through the utilization of blind gastric resection when indicated. As the number of patients so treated increases, this 100 per cent average must inevitably decrease, but when these encouraging results are compared to the high rate of recurrent and fatal hemorrhage following simple exploration without removal of the bleeding point, the procedure seems amply justified.

In doing a blind gastric resection, one should inspect carefully both the open duodenal stump and the gastric remnant to make certain that no bleeding points are neglected. If the source of hemorrhage is not removed in the resected specimen, the probability of recurrent hemorrhage is little altered by the gastrectomy. This rather obvious point was brought home to us by a recent experience with such a patient.

The patient was operated upon by one of us (LKF) to control a massive upper gastrointestinal hemorrhage which had recurred in the hospital on good conservative management. When no bleeding lesion was encountered, a high partial gastric resection was done according to the principles previously outlined. After thorough inspection of the specimen failed to reveal a mucosal erosion, recurrent hemorrhage was anticipated. This concern was justified 12 hours postoperatively when bleeding resumed. It soon became obvious that death would ensue unless the hemorrhage could be controlled surgically, and a second laparotomy was done 24 hours after the original resection. At this time fresh blood and large clots in the duodenum established this as the location of the bleeding point. A mucosal erosion was soon located in the second part of the duodenum and was resected. With his post-operative course uncomplicated by further hemorrhage, recovery was complete, and the patient is now alive and well.

Although this experience demonstrates that blind resection is not a panacea for upper gastrointestinal hemorrhage, it also reveals what can be accomplished by an aggressive approach to the problem. There is no doubt that this patient was salvaged by these surgical procedures. The blind gastric resection which seemed at first to have been of little or no help played its part in the eventual outcome. Without this preliminary procedure it is doubtful if the location of the bleeding lesion could have been identified at the second operation.

In summary, we believe that it is the responsibility of the surgeon to prevent patients with massive upper gastrointestinal hemorrhage from bleeding to death. If operation is undertaken for this purpose and no bleeding lesion is excised, the threat of repeated hemorrhage in the immediate postoperative period is great. If no bleeding point is apparent at operation, this threat can be avoided by a blind gastric resection in a high percentage of patients so treated. In the unusual patient (1 out of 7 in our experience) in which the source of bleeding is not found in the resected specimen, threat of recurrent hemorrhage is little altered by the gastrectomy, but even in such an instance the procedure may not be without benefit. An aggressive surgical attitude should be maintained if these patients are to be salvaged.

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BOOK REVIEWS

The editors of The American Surgeon will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.

Injuries of the Spinal Cord. By GEORGE E. PRATHER, M.D., F.A.C.S., Consulting Surgeon for Urology, Boston City Hospital; Head of the Department of Urology, Beth Israel Hospital, and FRANK H. MAYFIELD, M.D., F.A.C.S., Assistant Professor of Clinical Medicine, Springfield, Illinois, Charles C Thomas, 1953, 396 pages. $8.75.

This volume is a tribute to the vast improvement in the management of spinal cord injuries that has taken place during and since World War II. It consists of contributions from nine authorities, each concisely yet adequately presenting the problem from different points of view.

Not only are the usual view points of the neurosurgeon, orthopedist, and urologist well covered, but excellent chapters on nutrition, associated injuries, decubiti, general rehabilitation, and psychologic considerations are found.

The excellent bibliography at the end of the chapters on nutrition, urological aspects, and neurosurgical aspects make this monograph very valuable as a ready reference book. The urological aspects chapter has been previously published in The American Lecture Series.

The 125 illustrations add to the value of this volume to any physician who plays any part in the management of the paraplegic.

WILLIAM P. WILLIAMSON, M.D

Thoracic Surgery and Related Pathology. By GUSTAF E. LINDSKOG, B.S., M.A., M.D., F.A.C.S., WILLIAM H. CARMALT, Professor of Surgery, Yale University School of Medicine, Surgeon-in-Chief University Service, Grace-New Haven Community Hospital, and AVERILL A. LEIBOW, B.S., M.D., Professor of Pathology, Yale University School of Medicine, Pathologist-in-Chief University Service, Grace-New Haven Community Hospital. Appleton-Century-Crofts, Inc., New York, 1953. $15.00.

This book of more than 600 pages brings thoracic surgery up to date. It not only contains pathology related to thoracic surgery, but also related anatomy, embryology, and physiology. It is clearly written without any padding and is well illustrated.

Pulmonary tuberculosis is thoroughly discussed in 192 pages. The latest methods of diagnosis and treatment in such rapidly developing subjects as congenital anomalies of the respiratory and vascular systems are adequately presented. The use of antibiotics, postoperative complications and cardiac arrest receive proper emphasis. In fact, there are no weak chapters to be found. The literature on the subjects discussed is well reviewed and references appear at the end of each chapter.

Anyone doing thoracic surgery will appreciate the book as a ready reference. It should be available to all residents, interns, and students in surgery.

THOMAS G. ORR, M.D.

On Burns. Compiled and Edited by NATHAN A. WOMACK, M.D. Participants: DR. WILLIAM ALTEMEIER, DR. JAMES BARRETT BROWN, DR. W. J. H. BUTTERFIELD, DR. TRUMAN BLOCKER, DR. JULIAN BRUNER, DR. STUART CULLEN, DR. SAM MAY, DR. CARL MOYER, and DR. SIDNEY ZIFFREN. Charles C Thomas, Springfield, Illinois.

The participants in this symposium are all authorities in their fields. The book is not long, only 178 pages, and is one of the American Lecture Series publications. There are no illustrations. It contains a complete discussion of each subject with questions and answers as pre-

sented at a conference on burns at the University of Iowa Hospitals held in 1951. Practical points in the treatment of burned patients are covered in the detailed discussion. It is full of many controversial points ably discussed and defended by a panel of experts.

The essential problems of the patient as a whole, as regards fluids and electrolyte replacement, are covered by Dr. Carl Moyer. ACTH and cortisone, their dangers and abuses of usage, are well discussed by Dr. William Altemeier and Dr. W. J. H. Butterfield, respectively surgeon and internist. Pain and the use of narcotic drugs are carefully considered in a discussion led chiefly by Dr. Stuart Cullen. The merits of open versus the closed treatment of burns are presented and argued by Dr. Truman Blocker and Dr. James Barrett Brown, as well as by the other members of the panel. A consideration of the subject of surgical bacteriology of burns and problems in local care are participated in by the group led by Dr. William Altemeier. Dr. James Barrett Brown further discussed the coverage problem, and, as always, made important practical points based upon his vast knowledge in this field. The other members of the symposium, chiefly from the University of Iowa Medical Center, added a discussion of cases and presentation of patients treated at their hospital.

This reviewer has used the symposium as a definite teaching medium for his undergraduate and postgraduate students so that they may quickly—in one or two nights—obtain a good grasp of the fundamentals of burn care. This book is a most worthwhile addition to the library of anyone who has to treat a burned patient.

DAVID W. ROBINSON, M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Respiratory Diseases and Allergy. New Method of Approach. By JOSEF S. SMUL, M.D. Author of Digestive Diseases and Food Allergy. Fellow of National Gastro-Ent. Assoc.; Formerly: Vice President Manhattan Roentgen Ray Soc.; Assoc. Gastro-Ent. Beth David Hospital; Clin. Asst. Phys. Beth Israel Hospital, New York, Medical Library Company, 1953. $2.75.

Handbook of Differential Diagnosis. By HAROLD THOMAS HYMAN, M.D. Philadelphia, London and Montreal, J. B. Lippincott Company. $6.75.

Living with a Disability. By HOWARD A. RUSK, M.D., and EUGÉNE TAYLOR, in collaboration with MURIEL ZIMMERMANN, O.T.R. and JULIA JODSON, M.D. The Institute of Physical Medicine and Rehabilitation, New York Rehabilitation New York University-Bellevue Medical Center. Garden City, New York, The Blakiston Company, Inc., 1953. $3.50.

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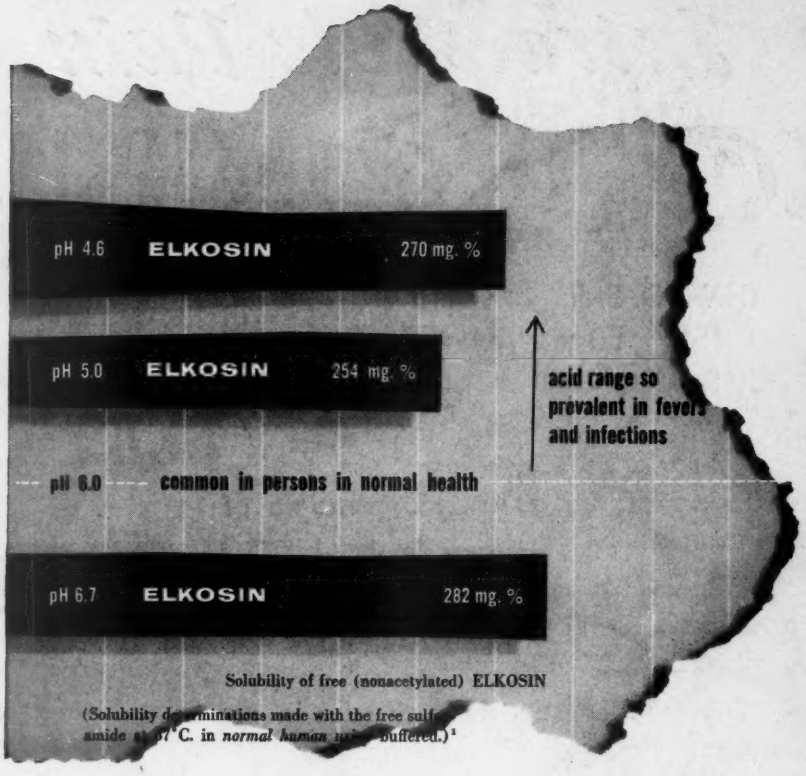
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